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NEWS 20	FEB 23	TOXCENTER updates mirror those of MEDLINE - more precise author group fields and 2009 MeSH terms
NEWS 21	FEB 23	Three million new patent records blast AEROSPACE into STN patent clusters
NEWS 22	FEB 25	USGENE enhanced with patent family and legal status display data from INPADOCDB
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=> s (GROWTH(W)HORMONE OR HUMAN(W)GROWTH(W)HORMONE OR GH OR HGH)
L1      254713 (GROWTH(W) HORMONE OR HUMAN(W) GROWTH(W) HORMONE OR GH OR HGH)

=> s l1 and Parkinson?
L2         727 L1 AND PARKINSON?

=> s l2 and py<2003
L3         480 L2 AND PY<2003

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4          280 DUP REM L3 (200 DUPLICATES REMOVED)

=> s l4 and (treat? or therap?)
L5         184 L4 AND (TREAT? OR THERAP?)

=> s l5 and (0.1 or 1 or 6 or 10 or mg or dosage)
L6          94 L5 AND (0.1 OR 1 OR 6 OR 10 OR MG OR DOSAGE)

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(FILE 'HOME' ENTERED AT 12:31:48 ON 02 APR 2009)

FILE 'MEDLINE, EMBASE, BIOSIS, CAPLUS' ENTERED AT 12:32:23 ON 02 APR 2009
L1 254713 S (GROWTH(W)HORMONE OR HUMAN(W)GROWTH(W)HORMONE OR GH OR HGH)
L2 727 S L1 AND PARKINSON?
L3 480 S L2 AND PY<2003
L4 280 DUP REM L3 (200 DUPLICATES REMOVED)
L5 184 S L4 AND (TREAT? OR THERAP?)
L6 94 S L5 AND (0.1 OR 1 OR 6 OR 10 OR MG OR DOSAGE)

=> dis ibib abs l6 1-94

L6 ANSWER 1 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1999216739 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10200732
TITLE: Predictors of treatment response from a first episode of schizophrenia or schizoaffective disorder.
AUTHOR: Robinson D G; Woerner M G; Alvir J M; Geisler S; Koreen A; Sheitman B; Chakos M; Mayerhoff D; Bilder R; Goldman R; Lieberman J A
CORPORATE SOURCE: Department of Psychiatry, Hillside Hospital, Long Island Jewish Medical Center, New Hyde Park, N.Y., USA.
CONTRACT NUMBER: MH-00537 (United States NIMH NIH HHS)
MH-41646 (United States NIMH NIH HHS)
MH-41960 (United States NIMH NIH HHS)
SOURCE: The American journal of psychiatry, (1999 Apr) Vol. 156, No. 4, pp. 544-9.
Journal code: 0370512. ISSN: 0002-953X.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
(COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199904
ENTRY DATE: Entered STN: 4 May 1999
Last Updated on STN: 4 May 1999
Entered Medline: 21 Apr 1999

AB OBJECTIVE: This study examined the treatment response of patients with first-episode schizophrenia and schizoaffective disorder and potential predictors of response. METHOD: First-episode patients were assessed on measures of psychopathology, cognition, social functioning, and biological parameters and treated according to a standardized algorithm. RESULTS: One hundred eighteen patients (52% male, mean age 25.2 years) entered the study. The cumulative percentage of patients responding by 1 year was 87%; the median time to response was 9 weeks. The following variables were significantly associated with less likelihood of response to treatment: male sex, obstetric complications, more severe hallucinations and delusions, poorer attention at baseline, and the development of parkinsonism during antipsychotic treatment. Variables not significantly related to treatment response were diagnosis (schizophrenia versus schizoaffective disorder), premorbid functioning, duration of psychotic symptoms prior to study entry, baseline disorganization, negative and depressive symptoms, baseline motor function, akathisia and dystonia during treatment, growth hormone and homovanillic acid measures, psychotic symptom activation to methylphenidate, and magnetic resonance measures. CONCLUSIONS: Patients with first-episode schizophrenia and schizoaffective disorder have high rates of response to antipsychotic treatment; there are specific

clinical and pathobiologic predictors of response.

L6 ANSWER 2 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1999142672 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9989566
TITLE: Clozapine and risperidone in chronic schizophrenia: effects on symptoms, parkinsonian side effects, and neuroendocrine response.
AUTHOR: Breier A F; Malhotra A K; Su T P; Pinals D A; Elman I; Adler C M; Lafargue R T; Clifton A; Pickar D
CORPORATE SOURCE: Experimental Therapeutics Branch, Division of Intramural Research Programs, NIMH, Bethesda, MD, USA.. breiervalan@lily.com
SOURCE: The American journal of psychiatry, (1999 Feb) Vol. 156, No. 2, pp. 294-8. Journal code: 0370512. ISSN: 0002-953X.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL) (COMPARATIVE STUDY) Journal; Article; (JOURNAL ARTICLE) (RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199902
ENTRY DATE: Entered STN: 11 Mar 1999 Last Updated on STN: 7 Apr 2000 Entered Medline: 25 Feb 1999

AB OBJECTIVE: Clozapine and risperidone were the first two "second-generation" antipsychotic drugs approved for schizophrenia. There is currently little information about their comparative efficacy from head-to-head clinical trials. The purpose of this study was to examine the comparative efficacy of clozapine and risperidone for positive and negative symptoms, depression, parkinsonian side effects, and indexes of neuroendocrine function in schizophrenic patients who met a priori criteria for partial response to traditional neuroleptic agents. METHOD: After a baseline fluphenazine treatment period, 29 patients participated in a 6-week, double-blind, parallel-group comparison of the effects of these agents. RESULTS: Clozapine was superior to risperidone for positive symptoms and parkinsonian side effects, but there were no significant differences between the drugs on two measures of negative symptoms, Brief Psychiatric Rating Scale total scores, and depression scores. The clozapine patients, but not the risperidone patients, demonstrated significant reductions from the fluphenazine baseline in positive symptoms, total symptoms, and depression. In addition, clozapine produced fewer effects on plasma prolactin than risperidone or fluphenazine. The mean daily doses during week 6 of the trial were 403.6 mg of clozapine and 5.9 mg of risperidone. CONCLUSIONS: The findings from this study indicate that these drugs have both important differences and similarities in their comparative efficacy in chronically ill, partially responsive patients with schizophrenia. Further research on second-generation antipsychotic drugs in this patient population that addresses key methodological issues, such as optimal dose and treatment duration, are needed.

L6 ANSWER 3 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1999136801 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9951625
TITLE: The nitric oxide hypothesis of aging.
AUTHOR: McCann S M; Licinio J; Wong M L; Yu W H; Karanth S; Rettorri V
CORPORATE SOURCE: Pennington Biomedical Research Center (LSU), Baton Rouge

70808-4124, USA.. mccannsm@mhs.pbrc.edu
CONTRACT NUMBER: DK43900 (United States NIDDK NIH HHS)
MH51853 (United States NIMH NIH HHS)
SOURCE: Experimental gerontology, (1998 Nov-Dec) Vol. 33, No. 7-8, pp. 813-26. Ref: 39
Journal code: 0047061. ISSN: 0531-5565.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199904
ENTRY DATE: Entered STN: 26 Apr 1999
Last Updated on STN: 26 Apr 1999
Entered Medline: 15 Apr 1999

AB Nitric oxide (NO), generated by endothelial (e) NO synthase (NOS) and neuronal (n) NOS, plays a ubiquitous role in the body in controlling the function of almost every, if not every, organ system. Bacterial and viral products, such as bacterial lipopolysaccharide (LPS), induce inducible (i) NOS synthesis that produces massive amounts of NO toxic to the invading viruses and bacteria, but also host cells by inactivation of enzymes leading to cell death. The actions of all forms of NOS are mediated not only by the free radical oxidant properties of this soluble gas, but also by its activation of guanylate cyclase (GC), leading to the production of cyclic guanosine monophosphate (cGMP) that mediates many of its physiological actions. In addition, NO activates cyclooxygenase and lipoxygenase, leading to the production of physiologically relevant quantities of prostaglandin E2 (PGE2) and leukotrienes. In the case of iNOS, the massive release of NO, PGE2, and leukotrienes produces toxic effects. Systemic injection of LPS causes induction of interleukin (IL)-1 beta mRNA followed by IL-beta synthesis that induces iNOS mRNA with a latency of two and four hours, respectively, in the anterior pituitary and pineal glands, meninges, and choroid plexus, regions outside the blood-brain barrier, and shortly thereafter, in hypothalamic regions, such as the temperature-regulating centers, paraventricular nucleus containing releasing and inhibiting hormone neurons, and the arcuate nucleus, a region containing these neurons and axons bound for the median eminence. We are currently determining if LPS similarly activates cytokine and iNOS production in the cardiovascular system and the gonads. Our hypothesis is that recurrent infections over the life span play a significant role in producing aging changes in all systems outside the blood-brain barrier via release of toxic quantities of NO. NO may be a major factor in the development of coronary heart disease (CHD). Considerable evidence has accrued indicating a role for infections in the induction of CHD and, indeed, patients treated with a tetracycline derivative had 10 times less complications of CHD than their controls. Stress, inflammation, and infection have all been shown to cause induction of iNOS in rats, and it is likely that this triad of events is very important in progression of coronary arteriosclerosis leading to coronary occlusion. Aging of the anterior pituitary and pineal with resultant decreased secretion of pituitary hormones and the pineal hormone, melatonin, respectively, may be caused by NO. The induction of iNOS in the temperature-regulating centers by infections may cause the decreased febrile response in the aged by loss of thermosensitive neurons. iNOS induction in the paraventricular nucleus may cause the decreased nocturnal secretion of growth hormone (GH) and prolactin that occurs with age, and its induction in the arcuate nucleus may destroy luteinizing hormone-releasing hormone (LHRH) neurons, thereby leading to decreased release of gonadotropins. Recurrent infections may play a role in aging of other parts of the brain, because there are increased numbers of astrocytes expressing IL-1 beta

throughout the brain in aged patients. IL-1 and products of NO activity accumulate around the plaques of Alzheimer's, and may play a role in the progression of the disease. Early onset Parkinsonism following flu encephalitis during World War I was possibly due to induction of iNOS in cells adjacent to substantia nigra dopaminergic neurons leading to death of these cells, which, coupled with ordinary aging fall out, led to Parkinsonism. The central nervous system (CNS) pathology in AIDS patients bears striking resemblance to aging changes, and may also be largely caused by the action of iNOS. Antioxidants, such as melatonin, vitamin C, and vitamin E, probably play an important acute and chronic role in reducing or eliminating the oxidant damage produced by NO.

L6 ANSWER 4 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1998446275 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9773098
TITLE: [Pharmacologic approach to autonomic failure]. Approche pharmacologique des dysautonomies.
AUTHOR: Senard J M; Montastruc J L
CORPORATE SOURCE: Laboratoire de Pharmacologie Medicale et Clinique, INSERM U 317, Faculte de Medecine, Toulouse, France.
SOURCE: Therapie, (1998 Jan-Feb) Vol. 53, No. 1, pp. 35-41. Ref: 80
Journal code: 0420544. ISSN: 0040-5957.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: French
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199811
ENTRY DATE: Entered STN: 6 Jan 1999
Last Updated on STN: 6 Jan 1999
Entered Medline: 3 Nov 1998
AB Four different forms of primary autonomic failure (multiple system atrophy, pure autonomic failure, Parkinson's disease and dopamine beta-hydroxylase deficiency) have been described. The first part of the article will focus on the interest to pharmacology of elucidating pathophysiological mechanisms underlying autonomic involvement at the central level (growth hormone response to clonidine acute challenge), presynaptic level (plasma catecholamine levels after yohimbine administration) and on post-synaptic receptors (binding studies, pressor responses to noradrenaline). The second part will discuss efficacy and side-effects of some of the many drugs which are currently proposed for the treatment of one of the most disabling symptoms related to autonomic failure, orthostatic hypotension. Special attention will be paid to drugs acting on blood composition (fludrocortisone, erythropoietin), on post-synaptic alpha-adrenoceptors (midodrine and clonidine) and on noradrenaline spill-over (yohimbine and L-Threo-DOPS).

L6 ANSWER 5 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1997360797 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9217760
TITLE: Distinction of idiopathic Parkinson's disease from multiple-system atrophy by stimulation of growth-hormone release with clonidine.
AUTHOR: Kimber J R; Watson L; Mathias C J
CORPORATE SOURCE: University Department of Clinical Neurology, National Hospital for Neurology and Neurosurgery/Institute of Neurology, London, UK.
SOURCE: Lancet, (1997 Jun 28) Vol. 349, No. 9069, pp. 1877-81.

Journal code: 2985213R. ISSN: 0140-6736.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199707
ENTRY DATE: Entered STN: 12 Aug 1997
Last Updated on STN: 12 Aug 1997
Entered Medline: 28 Jul 1997

AB BACKGROUND: Idiopathic Parkinson's disease is a common neurodegenerative disease that is difficult to distinguish from other parkinsonian syndromes such as multiple-system atrophy (MSA). In MSA, autonomic dysfunction is common and is associated with either parkinsonian or cerebellar features, or both. Differentiation of idiopathic Parkinson's disease from MSA is important because prognosis, complications, and response to therapy vary according to disorder. Our aim was to find out whether clonidine/growth hormone (GH) testing distinguishes idiopathic Parkinson's disease from MSA. METHODS: Clonidine is a centrally active alpha 2-adrenoceptor agonist that raises concentrations of GH in serum in healthy people and those with pure autonomic failure (with peripheral lesions), but not in those with MSA (with a central autonomic deficit). We investigated the effects of clonidine on 14 people with idiopathic Parkinson's disease (without autonomic deficits). 31 people with MSA of the three different clinical forms (parkinsonian, cerebellar, and mixed), 19 people with pure autonomic failure, and 27 healthy participants. In nine people with parkinsonian MSA (MSA-P), the GH response to levodopa was also assessed. FINDINGS: Clonidine raised serum GH concentrations in patients with idiopathic Parkinson's disease (median increase 8.98 [IQR 6.6-16.6] mU/L), normal participants (13.2 [7.0-18.6] mU/L), and patients with pure autonomic failure (12.5 [5.6-18.2] mU/L). In those with MSA who had central autonomic failure, GH concentrations were unchanged (MSA-P; 0.41 [-0.30 to 2.09] mU/L and cerebellar MSA [MSA-C] 1.67 [0-4.49] mU/L). The GH response to clonidine in idiopathic Parkinson's disease was significantly different from that in MSA-P ($p < 0.0002$). In MSA-P, the dopamine precursor levodopa raised GH concentrations (from mean 2.7 [SE 1.0] mU/L to mean 18.2 [6.0] mU/L, $p < 0.05$) and GH-releasing hormone (GHRH) concentrations (from mean 20.6 [3.25] ng/L to mean 68.0 [10.6] ng/L, $p < 0.05$), excluding dysfunction of pituitary somatotrophs or GHRH neurons as a cause for the absent GH response to clonidine in MSA. INTERPRETATION: The GH responses to clonidine clearly differentiated idiopathic Parkinson's disease from MSA-C and MSA-P. Together with the levodopa studies they indicated a specific alpha 2-adrenoceptor-hypothalamic deficit in MSA. The clonidine-GH test may provide further insight into central neurotransmitter and alpha 2-adrenoceptor-hypothalamic abnormalities in MSA.

L6 ANSWER 6 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1997251616 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9097298
TITLE: Defective 5-HT 1-receptor-mediated neurotransmission in the control of growth hormone secretion in Parkinson's disease.
AUTHOR: Volpi R; Caffarra P; Scaglioni A; Boni S; Saginario A; Chiodera P; Coiro V
CORPORATE SOURCE: Department of Internal Medicine, University of Parma,

Italy.
SOURCE: Neuropsychobiology, (1997) Vol. 35, No. 2, pp. 79-83.
Journal code: 7512895. ISSN: 0302-282X.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199707
ENTRY DATE: Entered STN: 24 Jul 1997
Last Updated on STN: 24 Jul 1997
Entered Medline: 16 Jul 1997

AB In order to gain a better insight in the serotonergic disorder affecting the parkinsonian brain, the growth hormone (GH) response to the 5-HT 1 serotonergic receptor agonist sumatriptan was tested. Sumatriptan was injected subcutaneously in 10 de novo parkinsonian patients (aged 58-69 years) and in 9 age-matched normal controls. On different occasions, subjects were also tested with GH-releasing hormone (GH-RH; 1 micrograms/kg body weight in an intravenous bolus) and L-arginine (30 g in 50 ml normal saline over 30 min), which releases GH from somatostatin inhibition, to determine whether GH secretion in response to alternate secretagogues is preserved in Parkinson's disease. In addition, a control test with the administration of normal saline instead of drug treatments was performed. Plasma GH levels were recorded over 2 h in all tests. Placebo administration did not change plasma GH levels in any subject. Similar GH responses were observed in normal controls and parkinsonian patients when GH-RH or arginine were administered. A significant GH increase was observed in normal controls after sumatriptan injection; in contrast, GH secretion was not modified by sumatriptan administration in parkinsonian patients. These data show that Parkinson's disease is associated with an impairment in the 5-HT1-receptor-mediated serotonergic transmission in the control of GH secretion, suggesting that this specific defect might alter other serotonergic-mediated mechanisms in the parkinsonian brain.

L6 ANSWER 7 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1997021160 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8867520
TITLE: Effects of terguride on anterior pituitary function in parkinsonian patients treated with L-dopa: a double-blind study versus placebo.
AUTHOR: Martignoni E; Horowski R; Liuzzi A; Costa A; Dallabonzana D; Cozzi R; Attanasio R; Rainer E; Nappi G
CORPORATE SOURCE: Department of Neurology III, Institute C. Mondino, University of Pavia, Italy.
SOURCE: Clinical neuropharmacology, (1996 Feb) Vol. 19, No. 1, pp. 72-80.
Journal code: 7607910. ISSN: 0362-5664.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199611
ENTRY DATE: Entered STN: 19 Dec 1996
Last Updated on STN: 19 Dec 1996
Entered Medline: 15 Nov 1996

AB In a randomized double-blind study, 20 parkinsonian patients

(suffering from the disease for 2-18 years), chronically treated with levodopa (500-750 mg/day for 0.5-12 years), received terguride (1 mg b.i.d.) or placebo for 4 weeks. Growth hormone (GH), prolactin (PRL), thyroid-stimulating hormone (TSH), and insulin-like growth factor (IGF-I) secretions were studied before and after the morning dose of levodopa (250 mg p.o.), both before and at the end of study period. At the beginning of the study, basal hormonal levels were within normal limits, and levodopa administration induced a significant suppression in PRL and TSH levels (both $p < 0.01$)) and a significant increase in GH ($p < 0.01$). The same results were observed at the end of the study period in the placebo group. Addition of terguride induced a significant suppression in basal PRL levels ($p < 0.01$), whereas levodopa-induced hormonal changes were unaffected. These data suggest that the hypothalamic dopaminergic function that controls anterior pituitary hormones is preserved in parkinsonian patients, regardless of both the duration of the disease and the long-term treatment with levodopa. The strong additional prolactin-lowering effect of terguride indicates long-lasting dopaminergic effects, as is already known from hyperprolactinemic conditions. The dopaminergic effects of levodopa on TSH, GH, and IGF-I secretion were unchanged by terguride treatment. The anti-dopaminergic effects of terguride observed in the motor system in animal studies, as well as in levodopa-induced dyskinesias in parkinsonian patients, could not be observed in the case of the dopaminergic control of anterior pituitary hormones under the conditions of this study.

L6 ANSWER 8 OF 94 MEDLINE on STN

ACCESSION NUMBER:	1996089316 MEDLINE
DOCUMENT NUMBER:	PubMed ID: 8534585
TITLE:	Initial clinical experiences with dopamine D2 receptor imaging by means of 2'-iodospiperone and single-photon emission computed tomography.
AUTHOR:	Yonekura Y; Saji H; Iwasaki Y; Tsuchida T; Fukuyama H; Shimatsu A; Iida Y; Magata Y; Konishi J; Yokoyama A; et al
CORPORATE SOURCE:	Biomedical Imaging Research Center, Fukui Medical School, Japan.
SOURCE:	Annals of nuclear medicine, (1995 Aug) Vol. 9, No. 3, pp. 131-6. Journal code: 8913398. ISSN: 0914-7187.
PUB. COUNTRY:	Japan
DOCUMENT TYPE:	(COMPARATIVE STUDY) Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE:	English
FILE SEGMENT:	Priority Journals
ENTRY MONTH:	199602
ENTRY DATE:	Entered STN: 21 Feb 1996 Last Updated on STN: 21 Feb 1996 Entered Medline: 5 Feb 1996

AB Dopamine D2 receptor imaging was performed with 123I labeled 2'-iodospiperone (2'-ISP) and single-photon emission computed tomography (SPECT) in 9 patients: 4 with idiopathic Parkinson's disease, 2 with parkinsonism, 1 with Wilson's disease and 2 with pituitary tumor, and the results were compared with the data for 9 normal subjects. Following an intravenous injection of 123I-2'-ISP, early (within 30 min) and late (between 2 and 4 hr) SPECT images were obtained by means of a multi-detector SPECT scanner or a rotating gamma camera. In normal subjects, early SPECT images demonstrated uniform distribution of radioactivity in the cerebral gray matter and cerebellum reflecting regional cerebral blood flow, whereas late SPECT images showed high radioactivity only in the basal ganglia. All the patients with

Parkinson's disease also demonstrated symmetrical basal ganglia uptake in the late SPECT images, but it was diminished in parkinsonism and Wilson's disease. One patient with a growth hormone-producing pituitary tumor had a positive uptake in the tumor. These preliminary clinical data demonstrated that 2'-ISP can be used for SPECT imaging of D2 dopamine receptors and may be of clinical value for the diagnosis and planning of the treatment of neurological diseases.

L6 ANSWER 9 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1992126189 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1772578
TITLE: Failure of the gamma-aminobutyric acid (GABA) derivative, baclofen, to stimulate growth hormone secretion in Parkinson's disease.
AUTHOR: Volpi R; Scaglioni A; Marcato A; Caffarra P; Rossi G; Caffarri G; Delsignore R; Chiodera P; Coiro V
CORPORATE SOURCE: Chair of Medical Clinic, University of Parma, Italy.
SOURCE: Journal of neural transmission. Parkinson's disease and dementia section, (1991) Vol. 3, No. 4, pp. 259-64.
Journal code: 8914371. ISSN: 0936-3076.
PUB. COUNTRY: Austria
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199203
ENTRY DATE: Entered STN: 22 Mar 1992
Last Updated on STN: 22 Mar 1992
Entered Medline: 5 Mar 1992
AB In order to evaluate whether the stimulating effect of GABA on growth hormone (GH) secretion changes in patients affected by Parkinson's disease, ten male parkinsonian patients and ten age matched normal controls were tested with the GABA derivative and GABAergic agent Baclofen (10 mg in a single oral administration at 09.00 h) (experimental test). In a different occasion, normal men and parkinsonian patients were tested with a placebo (control test). Basal GH levels were similar in normal controls and parkinsonian patients and remained unmodified during the control test. Plasma GH levels rose three times within 120 min after the administration of baclofen in the normal subjects. In contrast, plasma GH concentrations remained unmodified after baclofen treatment in the parkinsonian patients. In agreement with previous reports in the literature showing alterations of GABAergic neurotransmission in the parkinsonian brain, these data show a reduced GABAergic control of GH secretion in patients with Parkinson's disease.

L6 ANSWER 10 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1991233807 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1903236
TITLE: Hypothalamo-pituitary function and dopamine dependence in untreated parkinsonian patients.
AUTHOR: Cusimano G; Capriani C; Bonifati V; Meco G
CORPORATE SOURCE: Department of Neurological Sciences, University La Sapienza, Rome, Italy.
SOURCE: Acta neurologica Scandinavica, (1991 Mar) Vol. 83, No. 3, pp. 145-50.
Journal code: 0370336. ISSN: 0001-6314.
PUB. COUNTRY: Denmark

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199106
ENTRY DATE: Entered STN: 7 Jul 1991
Last Updated on STN: 7 Jul 1991
Entered Medline: 20 Jun 1991

AB Altered prolactin and thyrotropin responses to the TRH test in parkinsonian patients are held to indicate an impairment of the tubero-infundibular dopaminergic axis (TIDA). We correlated the plasmatic prolactin (PRL), thyrotropin (TSH) and somatotropin (GH) responses to TRH and bromocriptine + TRH of 12 parkinsonian patients, who had never received anti-parkinsonian drugs, with the severity, the duration, the age of onset and the dopamine-dependence of the motor symptomatology as indicated by the therapeutic response to a six-month oral treatment with bromocriptine. Patients with basal motor impairment over 9 on the Webster Rating Scale (WRS), those with duration of the disease over 24 months and those with onset earlier than 55 years of age showed lower PRL responses than the respectively matched subgroups. Patients showing a therapeutic motor improvement over 50% on the WRS (dopamine-dependent or "responder") showed lower PRL and TSH and higher GH responses than the non-responders. These findings suggest that the TIDA impairment described in Parkinson's disease develops along with the progressive course of the extrapyramidal involvement and is strictly correlated with the dopamine-dependence of the motor impairment.

L6 ANSWER 11 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1991228689 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2028709
TITLE: Effects of cytidine 5'-diphosphocholine administration on basal and growth hormone-releasing hormone-induced growth hormone secretion in elderly subjects.
AUTHOR: Ceda G P; Ceresini G; Denti L; Magnani D; Marchini L; Valenti G; Hoffman A R
CORPORATE SOURCE: Chair of Gerontology and Geriatrics, University of Parma, Italy.
SOURCE: Acta endocrinologica, (1991 May) Vol. 124, No. 5, pp. 516-20.
Journal code: 0370312. ISSN: 0001-5598.
PUB. COUNTRY: Denmark
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199106
ENTRY DATE: Entered STN: 30 Jun 1991
Last Updated on STN: 30 Jun 1991
Entered Medline: 13 Jun 1991

AB The basal and GH-releasing hormone-stimulated secretion of GH declines in the elderly. We tested the ability of cytidine 5'-diphosphocholine, a drug used in the treatment of stroke and Parkinson's disease, to alter GH secretion in 11 healthy elderly volunteers, aged 69-84. Each subject received an iv infusion of 2 g of cytidine 5'-diphosphocholine or normal saline. GHRH and TRH were also administered during cytidine 5'-diphosphocholine infusions. The infusion of cytidine 5'-diphosphocholine induced a 4-fold (p less than 0.05) increase in serum GH levels over basal values. A small increase in GH was seen after GHRH administration. However, the addition of GHRH to the cytidine 5'-diphosphocholine infusion resulted in

a GH response which was significantly greater than that seen after GHRH alone; the integrated concentration of GH was more than 2-fold greater in the cytidine 5'-diphosphocholine treated group (706.85 +/- 185.1 vs 248.9 +/- 61.4 micrograms.l-1 .(120 min)-1; p = 0.01). The PRL and TSH responses to TRH were not significantly affected by cytidine 5'-diphosphocholine infusion, indicating that dopaminergic mechanisms are not involved. These studies demonstrate that cytidine 5'-diphosphocholine can enhance basal and GHRH-stimulated GH release in the elderly, but the mechanism of action of the drug remains unclear.

L6 ANSWER 12 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1988263452 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3290992
TITLE: Apomorphine in the evaluation of dopaminergic function in man.
AUTHOR: Lal S
CORPORATE SOURCE: Department of Psychiatry, Montreal General Hospital.
SOURCE: Progress in neuro-psychopharmacology & biological psychiatry, (1988) Vol. 12, No. 2-3, pp. 117-64. Ref: 298
Journal code: 8211617. ISSN: 0278-5846.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T) General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198808
ENTRY DATE: Entered STN: 8 Mar 1990
Last Updated on STN: 8 Mar 1990
Entered Medline: 9 Aug 1988

AB 1. Apomorphine (Apo), a short acting dopamine (DA) receptor agonist, stimulates growth hormone (GH) secretion, decreases prolactin secretion, induces yawning, penile erections and other physiological effects in man. An effect on behavior, movement disorders and alcoholism has also been described. 2. Apo-mediated responses are used to evaluate DA function in psychiatric and neurological disorders. Many of the studies in schizophrenia using the GH response to Apo as an index of central DA function are difficult to interpret because of failure to control for key variables. 3. The GH response to Apo is a useful system to evaluate the effects of various drugs including peptides which may not cross the blood brain barrier on DA function in man. 4. Apo is a potent sedative. Specific antimanic, antischizophrenic, and anticraving effects in alcoholics have not been convincingly demonstrated. Side effects of Apo and failure to use active placebo make double-blind studies difficult. 5. Apo improves parkinsonian symptoms and certain forms of reflex epilepsy but beneficial effects in other involuntary movement disorders requires further documentation. 6. Apo may be a useful agent to evaluate DA function in impotent patients and predict a therapeutic response to long-acting dopaminergic agents. 7. Impairment of DA function may play a role in diabetic impotence. 8. The development of a simple polygraphic method to monitor the yawning response to Apo may facilitate clinical studies on the basic physiology of yawning in man and the use of the yawning response as a measure of central DA function in schizophrenia and other clinical disorders. 9. The use of Apo with 18F-fluorodeoxyglucose positron emission tomography to examine regional DA function in man opens up a promising area of research. 10. Though long-acting orally active aporphine DA agonists and antagonists have been developed the problem of tolerance may limit their therapeutic potential.

L6 ANSWER 13 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1988187691 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2965754
TITLE: Dopamine D-1 receptor agonist stimulation of prolactin secretion in man.
AUTHOR: Fabbrini G; Braun A; Mouradian M M; Tamminga C A; Chase T N
CORPORATE SOURCE: Experimental Therapeutics Branch, National Institute of Neurological and Communicative Disorders and Stroke, Bethesda, Maryland.
SOURCE: Journal of neural transmission, (1988) Vol. 71, No. 3, pp. 159-63.
Journal code: 0337042. ISSN: 0300-9564.
PUB. COUNTRY: Austria
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198805
ENTRY DATE: Entered STN: 8 Mar 1990
Last Updated on STN: 3 Mar 2000
Entered Medline: 26 May 1988
AB SKF 38393, a selective D-1 dopamine receptor agonist, elevated plasma prolactin levels in eight patients with various neurological disorders. Growth hormone concentrations were unaffected by SKF 38393 administration. The results suggest that D-1 receptors may be involved in the regulation of prolactin secretion.

L6 ANSWER 14 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1987204516 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2883678
TITLE: Clinical and neuroendocrine effects of zotepine--a new neuroleptic drug.
AUTHOR: von Bardeleben U; Benkert O; Holsboer F
SOURCE: Pharmacopsychiatry, (1987 Feb) Vol. 20, No. 1 Spec No, pp. 28-34.
Journal code: 8402938. ISSN: 0176-3679.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198706
ENTRY DATE: Entered STN: 3 Mar 1990
Last Updated on STN: 6 Feb 1995
Entered Medline: 9 Jun 1987
AB Zotepine, a new neuroleptic, was administered to 23 hospitalized patients with schizophrenia at doses of 75 to 600 mg/d for 21 to 42 days. Based upon analysis of conventional rating scales we observed a significant improvement (P less than 0.001) during week 1, which compound throughout the study period. After 21 days we identified 17 responders and 6 nonresponders, 2 of whom dropped out of the study because of a tonic-clonic seizure in one case and withdrawal of consent to further participation in the second case. During further treatment the improvement remaind stable in the responder group, while 1 nonresponder improved after 3 weeks of treatment . In 9 patients extrapyramidal symptoms were observed (6 parkinsonism, 2 early dyskinesia, 1 parkinsonism and early dyskinesia), which required sporadic (n = 3) or continuous (n = 2) treatment with biperiden in 5 cases. This low incidence of extrapyramidal symptoms necessitating coadministration of anticholinergic drugs suggests that the risk of inducing parkinsonism and dyskinesias during zotepine treatment is low. Comparison of

cortisol, growth hormone and prolactin release in normal controls challenged with 25 mg zotepine showed that only prolactin secretion is increased, while secretion of cortisol and growth hormone remains unaffected. The clinical effects observed in the present study show that zotepine has potential value in the treatment of schizophrenia. The findings warrant further study in controlled trials.

L6 ANSWER 15 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1987130802 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3815388
TITLE: Transdihydrolisuride in parkinsonism.
AUTHOR: Critchley P; Parkes D
SOURCE: Clinical neuropharmacology, (1987) Vol. 10, No. 1, pp. 57-64.
Journal code: 7607910. ISSN: 0362-5664.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198704
ENTRY DATE: Entered STN: 3 Mar 1990
Last Updated on STN: 3 Mar 1990
Entered Medline: 2 Apr 1987
AB The semisynthetic lisuride derivative transdihydrolisuride (terguride, TDHL) is an effective antiparkinsonian drug. In animals, TDHL appears to possess mixed dopamine agonist-antagonist effects, but this may not be the case in man. Single doses of TDHL were given to 21 subjects with parkinsonism. Overall, TDHL 0.25-0.5 mg caused dose-related improvement in parkinsonism for periods of up to 6 h, although 8 of 21 subjects showed no improvement or deterioration with TDHL 0.5-1 mg. In three patients with levodopa-induced psychosis, the addition of TDHL 0.75 mg daily for 5-10 days did not alter the psychotic state. In three subjects with levodopa-induced dyskinesias, the addition of TDHL 0.75 mg daily for 14 days resulted in a slight increase in the severity of involuntary movements. Side-effects of TDHL, sickness and hypotension, were similar to those observed with levodopa. Transdihydrolisuride caused prolonged inhibition of prolactin release, but unlike levodopa did not elevate plasma growth hormone levels. Additionally, TDHL did cause considerable sedation. These results may be due to combined effects of TDHL on nondopamine as well as dopamine neurotransmitter systems, rather than to partial or incomplete dopamine agonist effects.

L6 ANSWER 16 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1986191403 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3516579
TITLE: The safety of bromocriptine in long-term use: a review of the literature.
AUTHOR: Weil C
SOURCE: Current medical research and opinion, (1986) Vol. 10, No. 1, pp. 25-51. Ref: 196
Journal code: 0351014. ISSN: 0300-7995.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198605
ENTRY DATE: Entered STN: 21 Mar 1990

Last Updated on STN: 21 Mar 1990
Entered Medline: 28 May 1986

AB This paper reviews the safety data on bromocriptine administration for 1 to 10 years at daily doses of 1.25 to 80 mg in over 1100 patients with pituitary hormone overproduction (mainly from prolactinomas and growth-hormone producing adenomas), at daily doses of 3.75 to 170 mg in over 700 patients with Parkinson's disease, and at daily doses of 2.5 to 20 mg in 28 patients with various other conditions. In addition, information is provided on the safety for mother and child of bromocriptine administered at daily doses of 2.5 to 35 mg throughout gestation (54 pregnancies) or during its later stages (39 pregnancies). The side-effects of long-term bromocriptine treatment are usually no different from those seen during short-term treatment; most of them are relatively benign, and they have been shown in virtually all patients to be reversible. Bromocriptine appears to have no harmful effect on hepatic, renal, haematologic, or cardiac functions. It is considered that a hitherto unknown, severe though rare side-effect of bromocriptine is unlikely to be reported after such long experience.

L6 ANSWER 17 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1984137638 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6583312
TITLE: Dystonia--L-dopa responsive or juvenile parkinsonism?.
AUTHOR: Rondot P; Ziegler M
SOURCE: Journal of neural transmission. Supplementum, (1983) Vol. 19, pp. 273-81.
Journal code: 0425126. ISSN: 0303-6995.
PUB. COUNTRY: Austria
DOCUMENT TYPE: (CASE REPORTS)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198404
ENTRY DATE: Entered STN: 19 Mar 1990
Last Updated on STN: 19 Mar 1990
Entered Medline: 4 Apr 1984

AB Four cases of dystonia occurring in two families are reported. The first symptoms consisting of dystonia and rigidity appeared early in childhood, in the first months in one family and of ages two and five years respectively in the other. In two cases, transient tremor was noted. These four children have been treated with L-dopa with prompt spectacular results, in cases 1 and 2, with more gradual less complete results in the others. L-dopa treatment was continued twelve, eleven, six, and five years, respectively, without any developmental problems. Motor function remains satisfactory and school work is normal. The only secondary effect observed was the occurrence of dyskinesia. The relation between L-dopa responsive dystonia and Parkinson's disease is discussed.

L6 ANSWER 18 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1983186566 MEDLINE
DOCUMENT NUMBER: PubMed ID: 45469
TITLE: Effect of dopamine agonist (Lergotrile mesylate) therapy on twenty-four hour secretion of prolactin in treated Parkinson's disease.
AUTHOR: Bell R D; Carruth A; Rosenberg R N; Boyar R M
CONTRACT NUMBER: 1-K04 HD-00153 (United States NICHD NIH HHS)
5-M01-RR-00633 (United States NCRR NIH HHS)
HD-10909 (United States NICHD NIH HHS)

SOURCE: The Journal of clinical endocrinology and metabolism, (1978 Oct) Vol. 47, No. 4, pp. 807-11. Journal code: 0375362. ISSN: 0021-972X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T) (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198306
ENTRY DATE: Entered STN: 18 Mar 1990 Last Updated on STN: 3 Feb 1997 Entered Medline: 17 Jun 1983

AB Plasma PRL was measured at 20-min intervals in six patients with Parkinson's disease under various treatment protocols. In addition, 24-h mean GH levels were measured. The results of these studies showed that two untreated patients with Parkinson 's disease had normal 24-h mean PRL levels with the normal increase during sleep. During chronic treatment with L-dopa-carbidopa (Sinemet), the 24-h PRL level was 12.8 +/- 4.9 ng/ml (mean +/- SD) and there was persistence of augmented PRL secretion during sleep. The 24-h mean GH level ranged from 1.5-4.4 ng/ml, with a mean of 2.5 ng/ml. The addition of a dopamine agonist (Lergotrile mesylate) resulted in a significant (P less than 0.01) suppression of the 24-h mean PRL levels and abolition of the normal sleep augmentation after 2 weeks of therapy. This suppression was maintained in one patient who was restudied 4 months after the addition of dopamine agonist therapy to L-dopa-carbidopa. The 24-h mean GH levels did not change significantly after the addition of the dopamine agonist when compared to L-dopa-carbidopa alone. These results suggest a dichotomy between the PRL and GH responses to combined L-dopa-carbidopa and dopamine agonist therapy. In addition, the preservation of normal PRL regulation in the two untreated patients with Parkinson's disease suggests that dopaminergic neurons are not universally affected in this disorder.

L6 ANSWER 19 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1983186493 MEDLINE
DOCUMENT NUMBER: PubMed ID: 45463
TITLE: Effect of the dopamine agonist, lergotrile mesylate, on circulating anterior pituitary hormones in man.
AUTHOR: Thorner M O; Ryan S M; Wass J A; Jones A; Bouloux P; Williams S; Besser G M
SOURCE: The Journal of clinical endocrinology and metabolism, (1978 Aug) Vol. 47, No. 2, pp. 372-8. Journal code: 0375362. ISSN: 0021-972X.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL) (CONTROLLED CLINICAL TRIAL) Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198306
ENTRY DATE: Entered STN: 18 Mar 1990 Last Updated on STN: 3 Feb 1997 Entered Medline: 17 Jun 1983

AB The effects of the ergoline derivative, lergotrile mesylate, on the serum levels of PRL, GH, TSH, LH, FSH, cortisol, and blood sugar were studied in six normal males. The effects of lergotrile mesylate on the serum levels of GH and PRL were also studied in eight patients with acromegaly and in two with idiopathic hyperprolactinemia. In the

normal subjects, 2 mg oral lergotrile lowered basal PRL levels after 90 min and markedly impaired the PRL response to TRH (200 micrograms iv); the mean peak value +/- SE was 8.3 +/- 1.1 micrograms/liter, compared to the control value of 66.6 /+- 11.3 micrograms/liter. Lergotrile raised serum GH levels in five of the six subjects to peaks of 8-49 micrograms/liter, compared to 2-8 micrograms/liter after placebo. In three subjects, the GH response to lergotrile was attenuated by the prior administration of the dopamine antagonist, metoclopramide (10 mg orally). Lergotrile had no effect on FSH and LH levels under basal conditions or after the gonadotrophin-releasing hormone (GnRH; 100 micrograms iv). Circulating TSH levels were unaltered basally but impaired after TRH. Blood sugar levels were unaltered; serum cortisol was elevated in five of six subjects; there was a brief depression of diastolic blood pressure, but no change in pulse rate. The side effects after lergotrile were variable, with drowsiness as a consistent feature. These actions are similar to those of bromocriptine (an ergot derivative treatment of hyperprolactinemia and acromegaly, to suppress PRL and GH secretion, and in parkinsonism. Therefore, it may be expected that lergotrile could fulfill these clinical uses; however, in the studies comparing the effects of single oral doses of lergotrile (2 mg) and bromocriptine (2.5 mg) on GH and PRL secretion in patients with acromegaly and hyperprolactinemia, lergotrile in the dose used has been found to have an earlier onset and shorter duration of action.

L6 ANSWER 20 OF 94 MEDLINE on STN

ACCESSION NUMBER: 1983145374 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6131495
TITLE: Drug-induced growth hormone and prolactin responses in schizophrenia research.
AUTHOR: Lal S; Nair N P; Iskandar H I; Thavundayil J X; Etienne P; Wood P L; Guyda H
SOURCE: Progress in neuro-psychopharmacology & biological psychiatry, (1982) Vol. 6, No. 4-6, pp. 631-7. Journal code: 8211617. ISSN: 0278-5846.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198304
ENTRY DATE: Entered STN: 18 Mar 1990
Last Updated on STN: 6 Feb 1995
Entered Medline: 7 Apr 1983

AB 1. Interpretation of neuroendocrine studies in schizophrenia requires consideration of (a) the large number of variables that affect drug-induced endocrine responses (b) the effect of prior neuroleptic therapy (c) heterogeneity of schizophrenia (d) heterogeneity of receptors (e) uniqueness of the hypothalamic-pituitary axis (f) selectivity and pharmacokinetics of administered drugs. 2. Apomorphine increases growth hormone secretion by an effect on dopamine receptors that are not linked to adenylate cyclase and which are located outside the blood brain barrier. 3. Hypothalamic-pituitary histaminergic H2 and alpha-adrenergic function are unchanged in chronic schizophrenia. 4. Schizophrenic symptoms persist despite complete blockade of dopamine receptors modulating prolactin secretion. 5. Studies on dopamine receptors modulating prolactin secretion are unlikely to shed light on the pathophysiology of schizophrenia. 6. Screening for drugs which block apomorphine-induced growth hormone secretion but do not increase prolactin may provide a way of detecting anti-schizophrenic drugs which are devoid of side effects associated with hyperprolactinemia and which do not induce parkinsonism or

tardive dyskinesia.

L6 ANSWER 21 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1983015616 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6812112
TITLE: Neuroendocrine evidence for increased responsiveness of dopamine receptors in humans following electroconvulsive therapy.
AUTHOR: Balldin J; Granerus A K; Lindstedt G; Modigh K; Walinder J
SOURCE: Psychopharmacology, (1982) Vol. 76, No. 4, pp. 371-6.
Journal code: 7608025. ISSN: 0033-3158.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198212
ENTRY DATE: Entered STN: 17 Mar 1990
Last Updated on STN: 3 Mar 2000
Entered Medline: 2 Dec 1982
AB The previous finding that electroconvulsive therapy (ECT) enhances effects of dopamine (DA) agonists was further investigated in the present clinical experiment using neuroendocrine techniques. Apomorphine chloride (AP) (0.18-0.24 mg IV) induced stimulation of growth hormone (GH) and suppression of prolactin (PRL), as shown 2-3 days before and after ECT in mentally depressed patients (N = 12) and therapy-resistant parkinsonian patients with on-off phenomena (N = 9). AP-stimulated GH secretion was not significantly affected by ECT, whereas AP-induced suppression of PRL, expressed as percentage of baseline PRL levels, was significantly enhanced after ECT. Changes in clinical and hormonal parameters were not significantly correlated. Control patients not receiving ECT showed no significant changes in AP-induced GH secretion or PRL suppression in repeated investigations. The results support the view that ECT increases responsiveness of DA receptors and indicates that AP-induced suppression of PRL is a useful model to reflect these changes in humans.

L6 ANSWER 22 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1982218481 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7087591
TITLE: [Pharmacokinetic and pharmacodynamic aspects of L-DOPA treatment and dopadecarboxilase inhibitors and dopaminergic antagonists in Parkinson's disease (author's transl)].
Aspectos farmacocineticos y farmacodinamicos del tratamiento con L-DOPA + inhibidores de la DOPA descarboxilasa y agonistas dopaminergicos de la enfermedad de Parkinson.
AUTHOR: Garcia de Yebenes J; Avila C; Bazan E; Garcia E; Gervas J; Maseda C; Mena M; Muradas V; Ramos J A
SOURCE: Medicina clinica, (1982 Apr 1) Vol. 78, No. 7, pp. 259-64.
Journal code: 0376377. ISSN: 0025-7753.
PUB. COUNTRY: Spain
DOCUMENT TYPE: (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Spanish
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198208
ENTRY DATE: Entered STN: 17 Mar 1990

Last Updated on STN: 6 Feb 1998
Entered Medline: 26 Aug 1982

L6 ANSWER 23 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1982077100 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7310392
TITLE: Predictors for improvement after electroconvulsive therapy in parkinsonian patients with on-off symptoms.
AUTHOR: Balldin J; Granerus A K; Lindstedt G; Modigh K; Walinder J
SOURCE: Journal of neural transmission, (1981) Vol. 52, No. 3, pp. 199-211.
Journal code: 0337042. ISSN: 0300-9564.
PUB. COUNTRY: Austria
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198202
ENTRY DATE: Entered STN: 16 Mar 1990
Last Updated on STN: 16 Mar 1990
Entered Medline: 12 Feb 1982
AB The antiparkinsonian effect of electroconvulsive therapy (ECT) was investigated in nine parkinsonian patients with "on-off" phenomena. The patients were maintained on previously adjusted doses of antiparkinsonian drugs during and after ECT. Parkinsonian as well as mental symptoms were rated before and after treatment. Basal serum levels of prolactin (PRL) and growth hormone (GH) as well as apomorphine induced changes (0.24 mg i.v.) in these levels were investigated three days before start of treatment. Marked improvement of parkinsonian symptoms was seen in five patients. Two further patients showed slight improvements. The improvement persisted for 2-41 weeks. Improvement after ECT was found to correlate with age at the time of treatment and with duration of L-dopa therapy as well as the estimated life-dose of L-dopa. No correlation was found between depression before treatment, basal serum levels of GH and PRL or apomorphine induced changes in these hormone levels. The investigation indicates that ECT is a valuable adjuvant in the treatment of a selected group of parkinsonian patients with "on-off" phenomena. Furthermore, the results support our earlier proposal that ECT increases the responsiveness in postsynaptic dopamine sensitive structures.

L6 ANSWER 24 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1981177003 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6111843
TITLE: [Bromocriptine therapy]. Bromocriptintherapie.
AUTHOR: Konig M P
SOURCE: Schweizerische medizinische Wochenschrift, (1981 Feb 28) Vol. 111, No. 9, pp. 303-8.
Journal code: 0404401. ISSN: 0036-7672.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198106
ENTRY DATE: Entered STN: 16 Mar 1990
Last Updated on STN: 6 Feb 1995
Entered Medline: 23 Jun 1981
AB 1. The study of bromocriptine and its effects has opened up new perspectives on the highly sophisticated neuroendocrine control mechanisms

and the role of neurotransmitters. 2. As a specific prolactin inhibitor, bromocriptine is the treatment of choice in many cases of hyperprolactinemia in female and male. There is ample evidence that with bromocriptine a reduction of pituitary tumor size (particularly in prolactin-secreting tumors) can be achieved. 3. One highly specific use of bromocriptine, and which involves virtually no problems, is inhibition of puerperal lactation. 4. Bromocriptine is effective and useful in the treatment of acromegaly. While it may restore growth hormone levels to normal in mild and selective cases, it may be helpful in controlling severe cases in which surgical or radiotherapeutic approaches have failed to achieve satisfactory results. 5. As a dopamine agonist, bromocriptine offers a new possibility of treating parkinsonism. It may be given alone or, as is preferable in many cases, in combination with submaximal doses of levodopa. 6. Side effects are sometimes only observed on initiation of bromocriptine therapy, sometimes occur only during chronic therapy, and may occasionally necessitate interruption of the treatment. Sometimes continuation of therapy leads to tolerance of unwanted effects. Patients should be informed before the start of bromocriptine treatment about the possibility of side effects. With proper instruction on the manner in which the drug should be taken, many adverse reactions can be avoided or diminished.

L6 ANSWER 25 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1981158722 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7212665
TITLE: Lisuride in parkinsonism.
AUTHOR: Parkes J D; Schachter M; Marsden C D; Smith B; Wilson A
SOURCE: Annals of neurology, (1981 Jan) Vol. 9, No. 1, pp. 48-52.
Journal code: 7707449. ISSN: 0364-5134.
PUB. COUNTRY: United States
DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198105
ENTRY DATE: Entered STN: 16 Mar 1990
Last Updated on STN: 3 Mar 2000
Entered Medline: 13 May 1981
AB Lisuride is a soluble ergolene derivative with endocrine effects similar to but more potent than those of bromocriptine. In nine subjects with idiopathic, postencephalitic, or drug-induced parkinsonism, lisuride at a dosage of 0.05 to 0.15 mg intravenously caused an immediate improvement in tremor, rigidity, akinesia, and postural deformity, but also caused chorea and orofacial dyskinesia. Improvement lasted 2 to 3 hours. Lisuride had little or no effect in a single patient with progressively supranuclear palsy. Oral lisuride therapy, 0.8 to 4.8 mg daily, had similar effects but occasionally caused reduced awareness and hallucinations.

L6 ANSWER 26 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1981085316 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6108751
TITLE: gamma-Acetylenic GABA in tardive dyskinesia.
AUTHOR: Casey D E; Gerlach J; Magelund G; Christensen T R
CONTRACT NUMBER: 14081 (United States PHS HHS)
SOURCE: Archives of general psychiatry, (1980 Dec) Vol. 37, No. 12, pp. 1376-9.
Journal code: 0372435. ISSN: 0003-990X.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)

(CONTROLLED CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198102
ENTRY DATE: Entered STN: 16 Mar 1990
Last Updated on STN: 3 Mar 2000
Entered Medline: 24 Feb 1981

AB Brain gamma-aminobutyric acid (GABA) has been proposed to play a role in the modulation of extrapyramidal motor function. The effects of increasing brain GABA with gamma-acetylenic GABA (GAG), a drug that inhibits GABA transaminase, were evaluated in ten patients with stable tardive dyskinesia during a blind placebo-controlled trial. Drug effects during active treatment and two placebo periods were evaluated by scoring randomly sequenced videotapes of tardive dyskinesia and parkinsonian symptoms recorded weekly during a standardized examination. Tardive dyskinesia was significantly reduced, and preexisting parkinsonism increased slightly. The largest decrease in tardive dyskinesia symptoms occurred in patients receiving higher neuroleptic doses, suggesting an interaction between GABA and dopamine. Prolactin values increased but growth hormone values were unchanged. Psychiatric symptoms were also unchanged during GAG treatment.

L6 ANSWER 27 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1980254565 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7402308
TITLE: The role of D-1 and D-2 receptors.
AUTHOR: Schachter M; Bedard P; Debono A G; Jenner P; Marsden C D; Price P; Parkes J D; Keenan J; Smith B; Rosenthaler J; Horowski R; Dorow R
SOURCE: Nature, (1980 Jul 10) Vol. 286, No. 5769, pp. 157-9.
Journal code: 0410462. ISSN: 0028-0836.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198010
ENTRY DATE: Entered STN: 15 Mar 1990
Last Updated on STN: 6 Feb 1998
Entered Medline: 21 Oct 1980

AB Dopamine receptors in intracerebral motor and endocrine systems have been divided into two main types, D-1 and D-2, dependent on the presence or absence of adenylate cyclase linkage. Here we have investigated a number of dopamine agonist and antagonist drugs in man that have different actions on D-1 and D-2 receptors in animals. Motor and endocrine effects in parkinsonian subjects seem to depend on drug interaction with D-2, but not D-1, receptors. These results may have important implications for the design of anti-parkinsonian and antipsychotic agents.

L6 ANSWER 28 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1980100943 MEDLINE
DOCUMENT NUMBER: PubMed ID: 534183
TITLE: [Nycterohemeral variations of growth hormone and prolactin in 6 Parkinson's sufferers treated with bromocriptine (author's transl)].

TITLE: Variations nycthemerales de la somathormone et de la prolactine chez 6 parkinsoniens traites par bromocriptine.
AUTHOR: Passouant P; Besset A; Descomps B; Bonardet A; Billiard M; Negre C
SOURCE: La Nouvelle presse medicale, (1979 Oct 22) Vol. 8, No. 40, pp. 3237-42.
Journal code: 0312552. ISSN: 0301-1518.
PUB. COUNTRY: France
DOCUMENT TYPE: (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: French
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198003
ENTRY DATE: Entered STN: 15 Mar 1990
Last Updated on STN: 15 Mar 1990
Entered Medline: 17 Mar 1980

AB Secretions of GH and of PRL studied over a period of 24 hours in 6 untreated Parkinson's patients showed slight changes. The normal secretion of PRL in the female shows no nocturnal increase in the male. The secretion of GH linked to sleep is identified in the male and not in the female. These variations related to sex are interpreted as an increase in those normally found in the adult and facilitated by age. Bromocriptine given continuously at a dose of 10 to 20 mg/day for periods of 20 days to 6 months, results in suppression or a marked decrease in the 24-hour secretion of PRL. It has virtually no effect upon the secretion of GH. These results show that the dopaminergic regulation of PRL is preserved in Parkinson's disease.

L6 ANSWER 29 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1980013667 MEDLINE
DOCUMENT NUMBER: PubMed ID: 39308
TITLE: Failure of MIF-I to affect behavioral responses in patients with Parkinson's diseases under L-dopa therapy.
AUTHOR: Caraceni T; Parati E A; Girotti F; Celano I; Frigerio C; Cocchi D; Muller E E
SOURCE: Psychopharmacology, (1979 Jun 21) Vol. 63, No. 3, pp. 217-22.
Journal code: 7608025. ISSN: 0033-3158.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197911
ENTRY DATE: Entered STN: 15 Mar 1990
Last Updated on STN: 6 Feb 1995
Entered Medline: 28 Nov 1979

AB In eight subjects with Parkinson's disease under an optimal daily dose of L-dopa, acute administration of MIF-I (200 mg i.v.) did not ameliorate either the total disability score or the intellectual test PM 38 when evaluated in comparison with the effect induced by acute administration of a placebo. Also concomitant evaluation of the effect of MIF-I on the secretion of anterior pituitary hormones which are under dopaminergic control i.e., growth hormone and prolactin, did not reveal any potentiation of the L-dopa-induced stimulus.

L6 ANSWER 30 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1980011546 MEDLINE
DOCUMENT NUMBER: PubMed ID: 384250

TITLE: Drug therapy: Bromocriptine.
AUTHOR: Parkes D
SOURCE: The New England journal of medicine, (1979 Oct 18) Vol. 301, No. 16, pp. 873-8. Ref: 44
Journal code: 0255562. ISSN: 0028-4793.
PUB. COUNTRY: United States
DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 197911
ENTRY DATE: Entered STN: 15 Mar 1990
Last Updated on STN: 15 Mar 1990
Entered Medline: 28 Nov 1979

L6 ANSWER 31 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1979090181 MEDLINE
DOCUMENT NUMBER: PubMed ID: 104006
TITLE: Endocrine aspects of bromocriptine therapy in Parkinsonism.
AUTHOR: Shaw K M; Lees A J; Franks S; Daggett P; Thompson B D; Stern G M
SOURCE: Journal of neural transmission, (1978) Vol. 43, No. 2, pp. 153-60.
Journal code: 0337042. ISSN: 0300-9564.
PUB. COUNTRY: Austria
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197903
ENTRY DATE: Entered STN: 15 Mar 1990
Last Updated on STN: 15 Mar 1990
Entered Medline: 28 Mar 1979
AB Plasma growth hormone (GH) concentrations in Parkinsonian patients following 3 months optimum therapy with bromocriptine showed no significant change from pretreatment values, whilst plasma prolactin concentrations were uniformly suppressed. Pretreatment GH and prolactin levels were unrelated to clinical disability, and no correlation between hormonal changes and therapeutic response was found. These results suggest the presence of different dopaminergic receptor mechanisms for GH and prolactin release as well as between the extrapyramidal and neuroendocrine systems.

L6 ANSWER 32 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1979061513 MEDLINE
DOCUMENT NUMBER: PubMed ID: 363104
TITLE: Treatment of parkinsonism with N-n-propyl norapomorphine and levodopa (with or without carbidopa).
AUTHOR: Papavasiliou P S; Cotzias G C; Rosal V L; Miller S T
SOURCE: Archives of neurology, (1978 Dec) Vol. 35, No. 12, pp. 787-91.
Journal code: 0372436. ISSN: 0003-9942.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
(CONTROLLED CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 197901

ENTRY DATE: Entered STN: 14 Mar 1990
Last Updated on STN: 6 Feb 1998
Entered Medline: 26 Jan 1979

AB The effects of the concomitant administration of N-n-propyl norapomorphine (NPA) and levodopa, with and without carbidopa, were studied in 12 patients with unsatisfactory symptom control. Double-blind evaluation of the effects of NPA with suboptimal doses of levodopa or levodopa plus carbidopa (Sinemet) showed a mean overall improvement of 44% (20% to 74%) in nine patients and improvement of the "on-off" effect in five. Dyskinesia diminished in some patients after diminution of basal medication. In three patients, plasma dopa and growth hormone patterns did not differ substantially with and without NPA. The magnitude and timing of the therapeutic and side effects did not correlate with the pattern of growth hormone secretion, which suggests that this hormone might not be instrumental in the induction of these effects. N-n-propyl norapomorphine is a useful adjunct in the long-term management of patients with unsatisfactory response to levodopa.

L6 ANSWER 33 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1979021346 MEDLINE
DOCUMENT NUMBER: PubMed ID: 359016
TITLE: Plasma bromocriptine levels, clinical and growth hormone responses in Parkinsonism.
AUTHOR: Price P; Debono A; Parkes J D; Marsden C D; Rosenthaler J
SOURCE: British journal of clinical pharmacology, (1978 Oct) Vol. 6, No. 4, pp. 303-9.
Journal code: 7503323. ISSN: 0306-5251.
Report No.: NLM-PMC1429466.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197812
ENTRY DATE: Entered STN: 14 Mar 1990
Last Updated on STN: 14 Mar 1990
Entered Medline: 29 Dec 1978

AB 1. Plasma bromocriptine levels following separate oral doses of bromocriptine 12.5, 25, 50 and 100 mg have been determined in ten subjects with parkinsonism. 2. There was considerable variation between peak plasma bromocriptine levels in individual subjects after similar doses of bromocriptine. Peak levels occurred 30--210 min after dosage (mean 102 min). Peak clinical response, peak rise in plasma growth hormone level and fall in blood pressure followed shortly after peak bromocriptine levels occurred. 3. The shape of the plasma-time curve for bromocriptine was similar with all dosages. 4. There was no significant relationship between peak plasma bromocriptine levels, peak clinical response, peak increase in growth hormone and peak fall in blood pressure. However, the degree of improvement in the signs of parkinsonism was related to plasma bromocriptine levels was achieved. 5. Metoclopramide 60 mg pretreatment had no consistent effect upon plasma bromocriptine levels, the clinical or hormonal response.

L6 ANSWER 34 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1978148975 MEDLINE
DOCUMENT NUMBER: PubMed ID: 640244
TITLE: Effect of glucose on the glucagon response to L-dopa in normal and diabetic subjects.
AUTHOR: Klimes I; Vigas M; Jurcovicova J; Repcekova D; Kolesar P
SOURCE: Diabetes, (1978 Apr) Vol. 27, No. 4, pp. 396-9.

Journal code: 0372763. ISSN: 0012-1797.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 197806
ENTRY DATE: Entered STN: 14 Mar 1990
Last Updated on STN: 14 Mar 1990
Entered Medline: 17 Jun 1978

AB The effect of an oral dose of 1 gm. L-dopa either without or after a concomitant oral administration of 100 gm. glucose on the plasma level of pancreatic glucagon, plasma immunoreactive insulin (IRI), and plasma growth hormone (GH) was assessed in eight normal and 10 insulin-treated diabetic subjects. In the normal group the stimulatory effect o L-dopa on pancreatic glucagon release was reconfirmed. Moreover, in the diabetics essentially the same plasma glucagon increase after drug administration was found, such a response being inhibited in both groups by glucose. The increase of plasma GH after L-dopa in both healthy persons and diabetics and the inhibition of this response by glucose in healthy subjects was reconfirmed. Furthermore, the same effect of exogenous glucose on the L-dopa induced GH release was observed in diabetics. It may be concluded that glucagon may play a pathogenetic role in the worsening of parkinsonian diabetic patients during the treatment with L-dopa and that diabetic hyperglycemia per se seems to be insufficient for an inhibition of the release of both glucagon and GH AFTer L-dopa.

L6 ANSWER 35 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1977255358 MEDLINE
DOCUMENT NUMBER: PubMed ID: 578293
TITLE: Changes in pituitary hormones serum levels in bromocryptine-treated parkinsonian patients.
AUTHOR: Polleri A; Carolei A; Rolandi E; Masturzo P; Meco G; Agnoli A
SOURCE: Neuropsychobiology, (1977) Vol. 3, No. 1, pp. 42-8.
Journal code: 7512895. ISSN: 0302-282X.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197710
ENTRY DATE: Entered STN: 14 Mar 1990
Last Updated on STN: 14 Mar 1990
Entered Medline: 14 Oct 1977

AB In the course of a clinical trial with 2alpha-bromoergocryptin in Parkinson's disease, the serum levels of several pituitary hormones have been studied in the assumption that the drug active on nigro-striatal dopaminergic system might also interfere with hypothalamus-protuberantial neurotransmission, and have effects on the function of the pituitary. No changes in serum levels of FSH, LH, STH and TSH were detected for every dose of the drug employed. Only prolactin serum levels diminished since the beginning of the treatment, the decrease being significant (p less than 0.05 and p less than 0.01). This effect on prolactin does not change in the dose range considered. Clinical improvement was observed for doses of drugs above 15 mg /day, whereas the effect on prolactin secretion occurred with the dose of 7.5 mg/day.

L6 ANSWER 36 OF 94 MEDLINE on STN

ACCESSION NUMBER: 1977095989 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1036999
TITLE: Bromocriptine in Parkinsonism: long-term treatment, dose response, and comparison with levodopa.
AUTHOR: Parkes J D; Debono A G; Marsden C D
SOURCE: Journal of neurology, neurosurgery, and psychiatry, (1976 Nov) Vol. 39, No. 11, pp. 1101-8. Journal code: 2985191R. ISSN: 0022-3050. Report No.: NLM-PMC1083310.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (COMPARATIVE STUDY) Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197703
ENTRY DATE: Entered STN: 13 Mar 1990 Last Updated on STN: 13 Mar 1990 Entered Medline: 31 Mar 1977

AB Thirty-seven patients with Parkinsonism were treated with bromocriptine 2.5-300 mg daily. Bromocriptine, alone or combined with levodopa, caused a 20-30% reduction in disability scores in 11 patients treated for one year. Tolerance did not develop during this period. Bromocriptine treatment was not of value in six patients who had previously not responded or who had lost their response to levodopa. However, in four of five patients with response swings on levodopa due to rapid changes in plasma dopa levels, the addition of bromocriptine caused a more stable response. Dose response curves to bromocriptine 12.5, 25, 50, and 100 mg and to levodopa 250, 500, 1000, and 2000 mg were studied in seven patients. Levodopa 2 g had a greater therapeutic effect and caused a greater rise in plasma growth hormone concentration than bromocriptine 100 mg. Levodopa caused emesis more commonly and hallucinations less commonly than bromocriptine. Bromocriptine appears to be a less potent stimulant than dopamine, and has both pre- and post-synaptic effects. Metoclopramide 60 mg oral was given 30 minutes before bromocriptine or levodopa to establish whether this caused dopamine-receptor blockade. Metoclopramide acted as a competitive antagonist to the anti-Parkinsonism and growth hormone effect of both drugs and in individual cases prevented emesis and hallucinations. The fall in blood pressure due to bromocriptine or levodopa was not antagonised by metoclopramide. Central and peripheral vascular dopamine receptors may be different in nature.

L6 ANSWER 37 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1977030677 MEDLINE
DOCUMENT NUMBER: PubMed ID: 824407
TITLE: Weight loss in patients treated long-term with levodopa. Metabolic aspects.
AUTHOR: Vardi J; Oberman Z; Rabey I; Streifler M; Ayalon D; Herzberg M
SOURCE: Journal of the neurological sciences, (1976 Nov) Vol. 30, No. 1, pp. 33-40. Journal code: 0375403. ISSN: 0022-510X.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197701
ENTRY DATE: Entered STN: 13 Mar 1990 Last Updated on STN: 13 Mar 1990 Entered Medline: 3 Jan 1977

AB Seven aged Parkinsonian patients treated with levodopa (average dose 3-4 g daily for 1-3 years), showed a considerable weight loss. They were compared to two control groups of elderly and young volunteers after levodopa stimulation and after oral glucose tolerance tests. It was found that after levodopa administration the plasma free fatty acids, glucose, growth hormone and cortisol were significantly higher in the Parkinsonian group than in the young control group and only slightly higher than in the aged control group. It was also found that the serum insulin was significantly higher in Parkinsonian patients than in the aged control group. We think that the metabolic disturbances found in Parkinsonian patients are not solely due to levodopa administration but may be due to ageing processes. We suggest that weight loss in the older Parkinsonian patients treated over long periods with high doses of levodopa, is due to the enhancement of the lipolytic activity of the ageing fat cells caused by high levels of circulating insulin.

L6 ANSWER 38 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1976249841 MEDLINE
DOCUMENT NUMBER: PubMed ID: 942621
TITLE: Hereditary Parkinsonism-dystonia with sustained control by L-DOPA and anticholinergic medication.
AUTHOR: Allen N; Knopp W
SOURCE: Advances in neurology, (1976) Vol. 14, pp. 201-13.
Journal code: 0367524. ISSN: 0091-3952.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CASE REPORTS)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197609
ENTRY DATE: Entered STN: 13 Mar 1990
Last Updated on STN: 13 Mar 1990
Entered Medline: 25 Sep 1976

L6 ANSWER 39 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1976171046 MEDLINE
DOCUMENT NUMBER: PubMed ID: 772175
TITLE: Bromocriptine treatment in Parkinson's disease.
AUTHOR: Parkes J D; Marsden C D; Donaldson I; Galea-Debono A; Walters J; Kennedy G; Asselman P
SOURCE: Journal of neurology, neurosurgery, and psychiatry, (1976 Feb) Vol. 39, No. 2, pp. 184-93.
Journal code: 2985191R. ISSN: 0022-3050.
Report No.: NLM-PMC492245.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (CLINICAL TRIAL)
(CONTROLLED CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197607
ENTRY DATE: Entered STN: 13 Mar 1990
Last Updated on STN: 6 Feb 1998
Entered Medline: 6 Jul 1976
AB Thirty-one patients with Parkinson's disease were treated with the ergot alkaloid bromocriptine, a drug which stimulates dopamine receptors. Bromocriptine had a slight

therapeutic effect in patients on no other treatment and an additional effect in patients on levodopa. The mean optimum dosage of bromocriptine, established over a 12 week period, was 26 mg daily. In 20 patients bromocriptine was compared with placebo in a double-blind controlled trial. Active treatment caused a significant (P less than 0.02) reduction in total disability and akinesia scores. The least disabled patients showed the greatest response. Side-effects of bromocriptine--nausea, vomiting, hallucinations, and abnormal involuntary movements--were similar to nature to those of levodopa. In most normal subjects, bromocriptine causes an increase in plasma growth hormone concentration. This was determined in 20 patients with Parkinson's disease after 1-15 mg bromocriptine. Only a single patient showed an obvious increase up to 120 minutes after dosage. Bromocriptine was not effective treatment in two patients who had not previously responded to levodopa and replacement of this drug by bromocriptine in patients with end-of-dose akinesia after chronic levodopa treatment did not totally abolish response swings.

L6 ANSWER 40 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1976135204 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1252146
TITLE: Modification of the actions of some neuroactive drugs by growth hormone.
AUTHOR: Tang L C; Cotzias G C
SOURCE: Archives of neurology, (1976 Feb) Vol. 33, No. 2, pp. 131-4.
Journal code: 0372436. ISSN: 0003-9942.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 197604
ENTRY DATE: Entered STN: 13 Mar 1990
Last Updated on STN: 13 Mar 1990
Entered Medline: 9 Apr 1976
AB The flat serum growth hormone (GH) patterns of untreated parkinsonian patients develop diurnal rises during treatment with levodopa. This chronic exposure to excesses of GH might lead to the eventual emergence of the "on-off" phenomenon, which would indicate a need for animal experiments. Pretreatment of mice with GH increased (1) cerebral dopa and dopamine concentrations in levodopa-treated mice, (2) cerebral accumulation of injected tritiated apomorphine and tritiated thymidine, and (3) behavioral responses to levodopa, L-m-tyrosine, apomorphine hydrochloride, and oxotremorine.

L6 ANSWER 41 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1976100678 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1246301
TITLE: Letter: Somatostatin, growth hormone and parkinsonism.
AUTHOR: Cotzias G C; Papavasiliou P S
SOURCE: The New England journal of medicine, (1976 Feb 12) Vol. 294, No. 7, pp. 398.
Journal code: 0255562. ISSN: 0028-4793.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 197603

ENTRY DATE: Entered STN: 13 Mar 1990
Last Updated on STN: 13 Mar 1990
Entered Medline: 15 Mar 1976

L6 ANSWER 42 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1976100582 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1107835
TITLE: Treatment of Parkinson's disease with aporphines. Possible role of growth hormone.
AUTHOR: Cotzias G C; Papavasiliou P S; Tolosa E S; Mendez J S; Bell-Midura M
SOURCE: The New England journal of medicine, (1976 Mar 11) Vol. 294, No. 11, pp. 567-72. Journal code: 0255562. ISSN: 0028-4793.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL) Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.) (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 197604
ENTRY DATE: Entered STN: 13 Mar 1990
Last Updated on STN: 13 Mar 1990
Entered Medline: 2 Apr 1976

AB To avoid the main drawbacks of prolonged treatment with levodopa (involuntary movements and the "on-off" phenomenon), we administered apomorphine by mouth to 14 patients with Parkinson's disease. This treatment caused azotemia, which we circumvented by switching to N-propylnoraporpine, whose nephrotoxic dose (80 mg six times per day) was larger than its therapeutic dose (10 to 15 mg six times per day). Slowly increasing doses induced significant improvement (P less than 0.005) in all 24 patients studied, transitory mental aberrations in seven, and release of growth hormone in three patients tested. In patients previously on prolonged levodopa administration, the dyskinesia and "on-off" phenomenon were almost identical with N-propylnoraporphine, but both drawbacks were reduced or abolished in six patients by coadministration of alpha-methyldopa hydrazine plus levodopa. This coadministration seemed to abolish tachyphylaxis. We conclude that N-propylnoraporphine is very useful in the treatment of Parkinson's disease.

L6 ANSWER 43 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1976099377 MEDLINE
DOCUMENT NUMBER: PubMed ID: 54702
TITLE: Letter: Growth-hormone response to bromocriptine in parkinsonism.
AUTHOR: Shaw K M; Lees A J; Hayes S; Ross E J; Stern G M; Thompson B D
SOURCE: Lancet, (1976 Jan 24) Vol. 1, No. 7952, pp. 194. Journal code: 2985213R. ISSN: 0140-6736.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 197603
ENTRY DATE: Entered STN: 13 Mar 1990
Last Updated on STN: 13 Mar 1990
Entered Medline: 30 Mar 1976

L6 ANSWER 44 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1975114438 MEDLINE
DOCUMENT NUMBER: PubMed ID: 4452625
TITLE: Levodopa, manganese, and degenerations of the brain.
AUTHOR: Cotzias G C
SOURCE: Harvey lectures, (1974) Vol. 68, pp. 115-47.
Journal code: 0404252. ISSN: 0073-0874.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197506
ENTRY DATE: Entered STN: 10 Mar 1990
Last Updated on STN: 3 Feb 1997
Entered Medline: 13 Jun 1975

L6 ANSWER 45 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1975051517 MEDLINE
DOCUMENT NUMBER: PubMed ID: 4479702
TITLE: Decreased arginine-induced HGH response during L-dopa therapy in parkinsonian patients.
AUTHOR: Johnson S E; Norman N; Sjaastad O
SOURCE: Acta endocrinologica, (1974 Dec) Vol. 77, No. 4, pp. 686-90.
Journal code: 0370312. ISSN: 0001-5598.
PUB. COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197501
ENTRY DATE: Entered STN: 10 Mar 1990
Last Updated on STN: 10 Mar 1990
Entered Medline: 31 Jan 1975

L6 ANSWER 46 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1975045249 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1109209
TITLE: Protein intake and treatment of Parkinson 's disease with levodopa.
AUTHOR: Mena I; Cotzias G C
SOURCE: The New England journal of medicine, (1975 Jan 23) Vol. 292, No. 4, pp. 181-4.
Journal code: 0255562. ISSN: 0028-4793.
PUB. COUNTRY: United States
DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 197502
ENTRY DATE: Entered STN: 10 Mar 1990
Last Updated on STN: 10 Mar 1990
Entered Medline: 18 Feb 1975

L6 ANSWER 47 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1975029743 MEDLINE
DOCUMENT NUMBER: PubMed ID: 4371345
TITLE: Dissociation of growth hormone and

prolactin secretion in Parkinson's disease following chronic L-dopa therapy.
AUTHOR: Malarkey W B; Cyrus J; Paulson G W
SOURCE: The Journal of clinical endocrinology and metabolism, (1974 Aug) Vol. 39, No. 2, pp. 229-35. Journal code: 0375362. ISSN: 0021-972X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 197412
ENTRY DATE: Entered STN: 10 Mar 1990
Last Updated on STN: 10 Mar 1990
Entered Medline: 28 Dec 1974

L6 ANSWER 48 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1974165694 MEDLINE
DOCUMENT NUMBER: PubMed ID: 4133478
TITLE: Letter: Enhancement of levodopa-induced growth-hormone stimulation by propranolol.
AUTHOR: Camanni F; Massara F
SOURCE: Lancet, (1974 May 11) Vol. 1, No. 7863, pp. 942. Journal code: 2985213R. ISSN: 0140-6736.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 197407
ENTRY DATE: Entered STN: 10 Mar 1990
Last Updated on STN: 6 Feb 1998
Entered Medline: 16 Jul 1974

L6 ANSWER 49 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1974090067 MEDLINE
DOCUMENT NUMBER: PubMed ID: 4801258
TITLE: [Dopaminergic control of the diencephalo-pituitary axis for somatotropin secretion].
Controllo dopaminergico dell'asse diencefalo-ipofisario per la secrezione di ormone somatotropo.
AUTHOR: Cavagnini F; Pontiroli A E; Raggi U; Peracchi M; Malinverni A
SOURCE: Folia endocrinologica, (1973 Dec) Vol. 26, No. 6, pp. 483-9. Journal code: 0417431. ISSN: 0015-5535.
PUB. COUNTRY: Italy
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Italian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197403
ENTRY DATE: Entered STN: 10 Mar 1990
Last Updated on STN: 10 Mar 1990
Entered Medline: 31 Mar 1974

L6 ANSWER 50 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1973229424 MEDLINE
DOCUMENT NUMBER: PubMed ID: 4722572
TITLE: Plasma growth hormone and insulin response to levodopa and amantadine.
AUTHOR: Kytomaki O; Nousiainen R; Pekkarinen A; Rinne U K; Viljanen M
SOURCE: Journal of neural transmission, (1973) Vol. 34, No. 2, pp. 145-51.

Journal code: 0337042. ISSN: 0300-9564.
PUB. COUNTRY: Austria
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197310
ENTRY DATE: Entered STN: 10 Mar 1990
Last Updated on STN: 10 Mar 1990
Entered Medline: 9 Oct 1973

L6 ANSWER 51 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1973007806 MEDLINE
DOCUMENT NUMBER: PubMed ID: 5072652
TITLE: Blood levels of FSH, LH, TSH, and GH in parkinsonian patients before and during L-dopa treatment.
AUTHOR: Lundberg P O
SOURCE: Acta neurologica Scandinavica, (1972) Vol. 48, No. 4, pp. 427-32.
Journal code: 0370336. ISSN: 0001-6314.
PUB. COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197211
ENTRY DATE: Entered STN: 10 Mar 1990
Last Updated on STN: 10 Mar 1990
Entered Medline: 16 Nov 1972

L6 ANSWER 52 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1973005385 MEDLINE
DOCUMENT NUMBER: PubMed ID: 5071365
TITLE: Effect of L-dopa administration on growth hormone secretion in normal subjects and Parkinsonian patients.
AUTHOR: Cavagnini F; Peracchi M; Scotti G; Raggi U; Pontiroli A E; Bana R
SOURCE: The Journal of endocrinology, (1972 Sep) Vol. 54, No. 3, pp. 425-33.
Journal code: 0375363. ISSN: 0022-0795.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197211
ENTRY DATE: Entered STN: 10 Mar 1990
Last Updated on STN: 10 Mar 1990
Entered Medline: 16 Nov 1972

L6 ANSWER 53 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1972268767 MEDLINE
DOCUMENT NUMBER: PubMed ID: 5056733
TITLE: Metabolic responses to acute and chronic L-dopa administration in patients with parkinsonism.
AUTHOR: Sirtori C R; Bolme P; Azarnoff D L
SOURCE: The New England journal of medicine, (1972 Oct 12) Vol. 287, No. 15, pp. 729-33.
Journal code: 0255562. ISSN: 0028-4793.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 197210
ENTRY DATE: Entered STN: 10 Mar 1990
Last Updated on STN: 10 Mar 1990
Entered Medline: 25 Oct 1972

L6 ANSWER 54 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1972263861 MEDLINE
DOCUMENT NUMBER: PubMed ID: 5066290
TITLE: Effect of L-dopa on pituitary TSH and GH secretion in Parkinson's disease.
AUTHOR: Sakoda M; Kusaka T; Baba S; Shirakata S
SOURCE: Nippon Naibunpi Gakkai zasshi, (1972 Jul 20) Vol. 48, No. 4, pp. 241-4.
Journal code: 0413717. ISSN: 0029-0661.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Japanese
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197210
ENTRY DATE: Entered STN: 10 Mar 1990
Last Updated on STN: 10 Mar 1990
Entered Medline: 18 Oct 1972

L6 ANSWER 55 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1972087867 MEDLINE
DOCUMENT NUMBER: PubMed ID: 5061778
TITLE: The effect of L-dopa on plasma growth hormone, insulin, and thyroxine.
AUTHOR: Kansal P C; Buse J; Talbert O R; Buse M G
SOURCE: The Journal of clinical endocrinology and metabolism, (1972 Jan) Vol. 34, No. 1, pp. 99-105.
Journal code: 0375362. ISSN: 0021-972X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 197203
ENTRY DATE: Entered STN: 10 Mar 1990
Last Updated on STN: 10 Mar 1990
Entered Medline: 27 Mar 1972

L6 ANSWER 56 OF 94 MEDLINE on STN
ACCESSION NUMBER: 1971037433 MEDLINE
DOCUMENT NUMBER: PubMed ID: 5481776
TITLE: Stimulation of human-growth-hormone secretion by L-dopa.
AUTHOR: Boyd A E 3rd; Lebovitz H E; Pfeiffer J B
SOURCE: The New England journal of medicine, (1970 Dec 24) Vol. 283, No. 26, pp. 1425-9.
Journal code: 0255562. ISSN: 0028-4793.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 197101
ENTRY DATE: Entered STN: 1 Jan 1990
Last Updated on STN: 1 Jan 1990
Entered Medline: 13 Jan 1971

ACCESSION NUMBER: 2003401947 EMBASE

TITLE: JP Morgan Hambrecht & Quist: 3DP, Lundbeck & Serono: 7-10 January 2002, San Francisco, CA, USA.
AUTHOR: Worker, Charlotte (correspondence)
CORPORATE SOURCE: Current Drugs Ltd., Middlesex House, 34-42 Cleveland Street, London W1T 4LB, United Kingdom. charlotte.worker@current-drugs.com
SOURCE: IDrugs, (2002) Vol. 5, No. 2, pp. 124-128.
ISSN: 1369-7056 CODEN: IDRUFN
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Conference Article; (Conference paper)
FILE SEGMENT: 039 Pharmacy
038 Adverse Reactions Titles
037 Drug Literature Index
036 Health Policy, Economics and Management
032 Psychiatry
030 Clinical and Experimental Pharmacology
026 Immunology, Serology and Transplantation
025 Hematology
016 Cancer
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Oct 2003
Last Updated on STN: 23 Oct 2003

L6 ANSWER 58 OF 94 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2000368232 EMBASE
TITLE: The anorexia of aging.
AUTHOR: MacIntosh, Caroline; Chapman, Ian M
CORPORATE SOURCE: Department of Medicine, University of Adelaide, Royal Adelaide Hospital, Adelaide, SA, Australia.
AUTHOR: Morley, John E (correspondence)
CORPORATE SOURCE: Division of Geriatric Medicine, St. Louis University Health Sciences Center and Geriatric Research, Education and Clinical Center, St. Louis, MO, United States. morley@slu.edu
AUTHOR: Morley, John E (correspondence)
CORPORATE SOURCE: Division of Geriatric Medicine, Saint Louis University Health Sciences Center, 1402 S. Grand Boulevard, St. Louis, MO 63104, United States. morley@slu.edu
AUTHOR: Morley, John E (correspondence)
CORPORATE SOURCE: Division of Geriatric Medicine, Sint Louis University, Health Sciences Center, 1402 S. Grand Boulevard, St. Louis, MO 63104, United States. morley@slu.edu
SOURCE: Nutrition, (2000) Vol. 16, No. 10, pp. 983-995.
Refs: 263
ISSN: 0899-9007 CODEN: NUTRER
PUBLISHER IDENT.: S 0899-9007(00)00405-6
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article; (Conference paper)
FILE SEGMENT: 017 Public Health, Social Medicine and Epidemiology
002 Physiology
020 Gerontology and Geriatrics
029 Clinical and Experimental Biochemistry
037 Drug Literature Index
005 General Pathology and Pathological Anatomy
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Nov 2000
Last Updated on STN: 16 Nov 2000

L6 ANSWER 59 OF 94 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1999025645 EMBASE

TITLE: Peptides as drugs.
AUTHOR: Edwards, C.M.B. (correspondence); Cohen, M.A.; Bloom, S.R.
CORPORATE SOURCE: ICSM Endocrine Unit, Hammersmith Hospital, London, United Kingdom.
SOURCE: QJM - Monthly Journal of the Association of Physicians, (1999) Vol. 92, No. 1, pp. 1-4.
Refs: 24
ISSN: 0033-5622 CODEN: QMJPFH
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Editorial
FILE SEGMENT: 039 Pharmacy
037 Drug Literature Index
030 Clinical and Experimental Pharmacology
008 Neurology and Neurosurgery
003 Endocrinology
025 Hematology
016 Cancer
029 Clinical and Experimental Biochemistry
LANGUAGE: English
ENTRY DATE: Entered STN: 28 Jan 1999
Last Updated on STN: 28 Jan 1999

L6 ANSWER 60 OF 94 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1998093760 EMBASE
TITLE: Quinagolide in hyperprolactinaemia.
AUTHOR: Brownell, Judith (correspondence)
CORPORATE SOURCE: 1906 Mill Fern Drive SE, Mill Creek, WA 98012-5811, United States.
SOURCE: Reviews in Contemporary Pharmacotherapy, (1998) Vol. 9, No. 1, pp. 1-75.
Refs: 17
ISSN: 0954-8602 CODEN: RCPHFW
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 003 Endocrinology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 9 Apr 1998
Last Updated on STN: 9 Apr 1998

AB Hyperprolactinaemia, the most common hypothalamic-pituitary disorder confronting clinicians, is responsible for at least 25% of menstrual cycle disturbances that result in infertility and hypogonadism and up to 60% of cases of galactorrhoea. Prolactinomas comprise about 50% of all pituitary adenomas and 12-15% of all intracranial tumours. Most microadenomas (< 10 mm) and nontumoral hyperprolactinaemia are found in women, while macroadenomas (> 10 mm) occur equally often in men and women. Macroadenomas may expand, causing neurological as well as endocrinological disturbances. The aim of treatment is to lower serum prolactin levels and, if tumour is present, to reduce its size while preserving normal pituitary function. Medical therapy has become the treatment of choice, since the tumour recurrence rate after surgery is relatively high and radiotherapy, the second option, lowers prolactin very slowly and rarely results in normalization. Unique amongst pituitary hormones, normal levels of prolactin are maintained through negative feedback action of the neurotransmitter dopamine (DA), the hypothalamic prolactin releasing factor. The dopamine D(2) receptor on the pituitary lactotroph is specific to prolactin, hence, the use of dopaminomimetic drugs for treatment of hyperprolactinaemia. The standard compound and first prolactin-inhibiting drug to be developed was

the lysergic acid amide, bromocriptine. Literature reports showed that bromocriptine normalized prolactin levels in a mean 77% of hyperprolactinaemic women, restoring menses in 84%. In patients with macroadenomas, normal prolactin levels were reached in an average of 69% and mean tumour shrinkage of > 50% in up to 65% of patients. However, as many as 20% of patients do not tolerate bromocriptine and a comparable percentage are resistant to the drug. Other agents with pharmacological profiles similar to that of bromocriptine have therefore been introduced. These, also ergot-derived, include pergolide and metergoline representing the clavines, and lisuride and cabergoline of the amino-ergolines. As with bromocriptine, none of the compounds binds with absolute specificity to the dopamine receptor, and most act with similar potency on several other neurotransmitter systems. Quinagolide is a new chemical entity whose design combines the substituted quinoline segment of the ergolines with the linear benzo[g]quinoline segment of the prototypic dopamine agonist, apomorphine. The compound binds directly to the lactotroph D(2) receptor, decreasing the synthesis and release of prolactin by reducing its gene transcription through its action on cyclic AMP. Quinagolide showed no action on adrenergic or serotoninergic receptors, and its oral and parenteral activity was up to 200-fold that of bromocriptine. The compound is rapidly and well absorbed, extensively metabolized and over 95% excreted in urine and faeces. The elimination half-lives of parent drug and metabolites are 22.3 h and 17.5 h, respectively. Studies in healthy individuals and in hyperprolactinaemic patients showed that maximal prolactin suppression was reached after 2-4 h and that single doses above 0.04 mg suppressed serum prolactin for 24 h. In addition, quinagolide was without negative effects on other hormones of the pituitary-thyroid, -adrenal, or -gonadal axes, or on growth hormone, and no influence was seen on plasma renin activity or aldosterone levels, both D(2) receptor controlled. Double-blind comparison with bromocriptine in 279 hyperprolactinaemic women resulted in normoprolactinaemia in 66% and 76% of the bromocriptine and quinagolide groups, respectively. Regular menses were restored in 82% of the women treated with bromocriptine and in 88% of those treated with quinagolide. Galactorrhoea was relieved in 95% of women in both groups. Adverse events were more frequent in the bromocriptine group, accounting for a significantly higher discontinuation rate in these women ($p < 0.01$). A review of quinagolide therapy in 603 women with idiopathic or microadenomatous hyperprolactinaemia treated in various series revealed a prolactin normalization rate of 87%. In 92.2% of patients normal menses were restored, and galactorrhoea disappeared in 91%. The collective results of treatment with quinagolide in 338 patients with macroadenomas showed that prolactin normalized in 74%; menses were established in 79% of women, and improved libido and sexual function were achieved in 88% of men. Galactorrhoea ceased in 95% of both male and female patients. Assessment of pituitary imaging showed that the tumours of 73% of the patients decreased in size by a mean 70% during quinagolide treatment, reaching maximal shrinkage within the first 6 months. In newly-diagnosed patients, maximal size decrease was documented in 76% of the tumours after 2 months of treatment. Visual field defects normalized in up to 84% of patients. Overall, quinagolide treatment resulted in normoprolactinaemia in 53% of bromocriptine-resistant and in 93% of bromocriptine-intolerant patients. Positive results were reported with the use of quinagolide in nonfunctioning pituitary adenomas, in puerperal lactation inhibition, in patients with acromegaly, and in Parkinson's disease. The major adverse events documented during treatment with quinagolide include nausea, headache, dizziness, and fatigue. The tolerability of the drug was judged by 91% of 670 patients to have been good or very good. Up to 40% of the patients spontaneously reported enhanced well-being. Fewer than 5% discontinued treatment because of adverse events. No electrocardiographic or

laboratory safety measures were adversely affected by quinagolide treatment. A normalizing trend was apparent in lipid levels and in body weights, particularly in prolactinoma patients with above-normal baseline values.

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ACCESSION NUMBER: 1995170916 EMBASE
TITLE: A dopaminergic hypothesis of major depression.
AUTHOR: Mann, J.J., Dr. (correspondence); Kapur, S.; Schatzberg, A.F.; Schwartz, J.-C.; Willner, P.
CORPORATE SOURCE: Department of Neuroscience, New York State Psychiatric Institute, Box 28, 722 W. 168th Street, New York, NY 10032, United States.
SOURCE: Clinical Neuropharmacology, (1995) Vol. 18, No. SUPPL. 1, pp. S57-S65.
ISSN: 0362-5664 CODEN: CLNEDB
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article; (Conference paper)
FILE SEGMENT: 032 Psychiatry
037 Drug Literature Index
038 Adverse Reactions Titles
005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 27 Jun 1995
Last Updated on STN: 27 Jun 1995

AB The dopaminergic system appears to play a role in the etiopathogenesis of major depression that is analogous to the roles hypothesized for norepinephrine and serotonin. Three distinct groups of dopaminergic neurons project via the nigrostriatal, mesolimbic, and mesocortical pathways, and they are involved in motor functioning, major depression, cognition, and a variety of behaviors related to reward and motivation. The five dopamine- receptor subtypes provide an additional level of organization of the dopaminergic system; medications that are direct agonists or antagonists for specific receptors will have more selective effects within the dopaminergic system. A variety of studies in animals, as well as clinical observations, are consistent with a dopamine-deficiency hypothesis of major depression. Depletion of dopamine levels by drugs such as reserpine and tetrabenazine, or through the long-term use of stimulants, has been reported to produce major depressive episodes in vulnerable individuals. The association of depression with Parkinson's disease provides important additional support for the dopaminergic hypothesis of depression. Electroconvulsive therapy , which enhances dopaminergic transmission, improves both depression and the motor symptoms of Parkinson's disease. The development of more selective medications will help to clarify the precise role of the dopaminergic system and specific receptor subtypes in the etiopathogenesis of major depression.

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ACCESSION NUMBER: 1994364887 EMBASE
TITLE: Drug-Induced dementia. Incidence, management and prevention.
AUTHOR: Starr, J.M.; Whalley, L.J., Prof. (correspondence)
CORPORATE SOURCE: Department of Mental Health, University Medical Buildings, Foresterhill, Aberdeen AB9 2ZD, United Kingdom.
SOURCE: Drug Safety, (1994) Vol. 11, No. 5, pp. 310-317.
ISSN: 0114-5916 CODEN: DRSAEA
COUNTRY: New Zealand

DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 21 Dec 1994
Last Updated on STN: 21 Dec 1994

AB Drugs are a frequently cited cause of dementia. There is a paucity of data regarding the incidence of drug-induced dementia, but it has been estimated that over 10% of patients attending memory clinics have iatrogcnic disease. Drugs may impair cognition indirectly via metabolic effects, such as hypoglycaemia, by alterations of immunological factors within the CNS, and by actions that interfere with synaptic transmission. Classes of drugs most frequently responsible are the benzodiazepines, antihypertensives and drugs with anticholinergic properties. Each of these classes is likely to produce a different pattern of neuropsychological deficits. Prevention of drug-induced dementia will be aided by: (i) minimising the number of drugs prescribed; (ii) using shorter-acting, preparations; (iii) avoiding agents that cross the blood-brain barrier where possible: (iv) evaluating renal and hepatic function regularly; and (v) briefly assessing cognitive function before treatment.

L6 ANSWER 63 OF 94 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1992330666 EMBASE
TITLE: Transplantation in the treatment of paralysis agitans (Parkinson's disease).
AUTHOR: Fazzini, E., Dr. (correspondence)
CORPORATE SOURCE: 650 First Ave, New York, NY 10016, United States.
SOURCE: Journal of the American Osteopathic Association, (1992) Vol. 92, No. 10, pp. 1255-1260.
ISSN: 0098-6151 CODEN: JAOAAZ
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 037 Drug Literature Index
008 Neurology and Neurosurgery
009 Surgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 29 Nov 1992
Last Updated on STN: 29 Nov 1992

AB Over the past 3 years, there has been great interest in transplantation therapy in the treatment of Parkinson's disease. Following the impressive results reported by Madrazo in the spring of 1987, more than 350 cases of adrenal medullary implantation have been performed worldwide. There has been a significant reduction in 'off' time and an increase in 'on' time without chorea in 40% of patients having this procedure. The duration of effect is 1 year in half of these cases, with the other half (20% of all patients) still demonstrating significant improvement 3 years after the procedure. The mechanism of the bilateral beneficial improvement is unknown. The survival of adrenal medullary tissue has not been demonstrated at autopsy. It is thought that the mechanism of improvement involves either regenerative sprouting of the remaining dopamine producing neurons as a consequence of the release of neurotrophic factors or an interruption of the striatal pallidal output inhibitory influence of the basal ganglia on the thalamus (or both). Fetal mesencephalic implantation has also been attempted in more than 100 cases worldwide. The improvements when seen are not any more dramatic than those following the best results of adrenal medullary implantation.

Graft survival has not been proved; it remains a possibility that interruption of the putaminosubthalamic pallidal pathway or a trophic influence of the tissue provides an alleviation in parkinsonism. The ethical controversy, need for long-term immunosuppression, and difficulty with obtaining tissue of the appropriate age and delivering the appropriate quantity to the putamen have made this technique less than adequate. Newer techniques employing genetic engineering, cultures of adrenal medullary tissue, and encapsulated xenografts are being investigated.

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ACCESSION NUMBER: 1990242463 EMBASE
TITLE: Neuroendocrinological effects of L-threo-3, 4-dihydroxyphenylserine (DOPS), a putative norepinephrine precursor, on healthy volunteers.
AUTHOR: Semba, J.; Nankai, M.; Okubo, Y.; Kato, M.; Matsuura, M.; Takahashi, R.
CORPORATE SOURCE: Department of Neuropsychiatry, Faculty of Medicine, Tokyo Medical and Dental University, Tokyo, Japan.
SOURCE: Japanese Journal of Psychiatry and Neurology, (1990) Vol. 44, No. 1, pp. 73-78.
ISSN: 0912-2036 CODEN: JJPNEA
COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
030 Clinical and Experimental Pharmacology
032 Psychiatry
037 Drug Literature Index
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 13 Dec 1991
Last Updated on STN: 13 Dec 1991

AB The effect of L-threo-3, 4-dihydroxyphenylserine (DOPS) on plasma cortisol, prolactin, thyrotropin-stimulating hormone (TSH) and growth hormone concentrations was studied in nine healthy male volunteers. The drug was administered orally (300 mg or 600 mg DOPS) using a multiple crossover placebo-controlled study design. Plasma hormone concentrations were measured at 30 minute intervals for 3 hours after dosing. Plasma DOPS peak concentrations were observed between 2 and 3 hours after dosing. DOPS, however, had no effect on plasma hormone concentrations and this may be attributed to the known low brain permeability of DOPS in healthy subjects.

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ACCESSION NUMBER: 1978282207 EMBASE
TITLE: [Bromocriptine in Parkinsonism. A clinical and biological study in 38 patients].
BROMOCRIPTINE ET MALADIE DE PARKINSON. ETUDE CLINIQUE ET BIOLOGIQUE CHEZ 38 MALADES.
AUTHOR: Schott, B.; Fischer, C.
CORPORATE SOURCE: Hop. Neurol., Lyon, France.
SOURCE: Lyon Medical, (1978) Vol. 239, No. 3, pp. 137-140.
ISSN: 0024-7790 CODEN: LYMEAN
COUNTRY: France
DOCUMENT TYPE: Journal
FILE SEGMENT: 020 Gerontology and Geriatrics
003 Endocrinology
037 Drug Literature Index
008 Neurology and Neurosurgery

LANGUAGE: French
SUMMARY LANGUAGE: English
AB 38 patients suffering from Parkinson's disease were treated with bromocriptine over an average period of 6 mth, with a daily dosage of 25.5 mg. Tolerance was good, with few adverse reactions. No correlation was found between growth hormone levels on bromocriptine and treatment effectiveness. Prolactin secretion decreased as in normal patients. Patients with no previous treatment had very good results. Failures previously treated with DOPA were also failures with bromocriptine, but in cases with decreased DOPA activity good results with bromocriptine can be achieved. In addition, the side effects of DOPA can be reduced. The efficacy of bromocriptine can be termed 'little DOPA'. Its better therapeutic indications seem to be: decrease of DOPA activity in patients with no mental deterioration, and attenuation of adverse reactions of prolonged DOPA-therapy.

L6 ANSWER 66 OF 94 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1977205132 EMBASE
TITLE: Bromocriptine and dopamine receptor stimulation.
AUTHOR: Debono, A.G.; Marsden, C.D.; Asselman andParkes, P.J.D.
CORPORATE SOURCE: Univ. Dept. Neurol., King's Coll. Hosp., London, United Kingdom.
SOURCE: British Journal of Clinical Pharmacology, (1976) Vol. 3, No. 6, pp. 977-982.
ISSN: 0306-5251 CODEN: BCPHBM
DOCUMENT TYPE: Journal
FILE SEGMENT: 030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
AB The response to different doses of bromocriptine (12.5, 25, 50 and 100 mg) has been established in six patients with Parkinson's disease. Bromocriptine, like levodopa, causes improved mobility in patients with Parkinsonism, emesis, hallucinations, a fall in supine and erect blood pressure, increase of plasma growth hormone and suppression of prolactin concentration. Bromocriptine (50 or 100 mg) has as great an anti Parkinsonian effect as average therapeutic doses of levodopa, and a longer duration of action, 6-10 hours. In the dose range studied, bromocriptine appears to be a complete dopamine agonist, although 100 mg was less effective than 50 mg in two patients. The different actions of bromocriptine and other dopamine agonist drugs may result from stimulation of different types of dopamine receptor.

L6 ANSWER 67 OF 94 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1976050953 EMBASE
TITLE: Bromocriptine.
SOURCE: Lancet, (1975) Vol. 1, No. 7915, pp. 1076-1077.
ISSN: 0140-6736 CODEN: LANCAO
DOCUMENT TYPE: Journal
FILE SEGMENT: 003 Endocrinology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
AB In a search for ergot derivatives that stimulate the release of prolactin inhibitory factor, Fluckiger reported that bromocriptine (2 bromo α ergokryptine; CB 154) seemed to be potent and was well tolerated. It was shown that bromocriptine was a dopamine agonist (i.e., bromocriptine activated dopaminergic postsynaptic receptors). The first clinical use for bromocriptine was suppression of puerperal lactation. A double blind

study of 60 patients showed that bromocriptine was as effective as stilbestrol. Bromocriptine was also found useful in the treatment of galactorrhea (non puerperal lactation). The therapeutic action of bromocriptine was associated in both conditions with a reduction of the raised plasma prolactin concentrations. There has been interest in the use of bromocriptine for treating acromegaly. Levodopa, presumably via dopaminergic pathways, is known to raise the plasma level of growth hormone in normal subjects, but in acromegaly it induces a paradoxical reduction of growth hormone. In 2 investigations encouraging results have been obtained with bromocriptine in acromegaly. There was clinical improvement and the plasma concentration of growth hormone fell. Bromocriptine was investigated in a neurological disorder associated with decreased dopaminergic transmission, idiopathic parkinsonism. A double blind trial showed that bromocriptine, added to the most effective dosage of antiparkinsonian drugs (mostly including levodopa), led to some 10% improvement in mildly affected patients and to almost 20% improvement in severely disabled patients.

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ACCESSION NUMBER: 1975190678 EMBASE
TITLE: Protein intake and treatment of Parkinson 's disease with levodopa.
AUTHOR: Mena, I.; Cotzias, G.C.
CORPORATE SOURCE: Med. Res. Cent., Brookhaven Nat. Lab., Upton, L.I., N.Y. 11973, United States.
SOURCE: New England Journal of Medicine, (1975) Vol. 292, No. 3, pp. 181-184.
ISSN: 0028-4793 CODEN: NEJMAG
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 020 Gerontology and Geriatrics
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
008 Neurology and Neurosurgery
LANGUAGE: English

AB The influence of protein ingestion on the therapeutic efficacy and metabolic effects of levodopa in Parkinson's disease was studied. Among 8 patients, differing in symptomatic control, neurologic scores (normal 0, maximal 100) on 2 g of protein per kg of body weight were, at 8 a.m., 27.8 ± 2.1 (mean±S.E.M.) and at 3 p.m., 46.7±2.6 ($p<0.001$). On 10 g of protein per day scores were 24.6±2.1 at 8 a.m. and 24.1±2.7 at 3 p.m. In 7 patients maintained on 0.5 g of protein per kg of body weight per day for 2 mth to 1 yr, levodopa requirements diminished progressively. Measurement of growth hormone in 5 patients off levodopa showed low constant levels without the normal fluctuations. Near normal patterns were found in 6 patients on levodopa, but tended to flatten out in 6 patients also taking a high protein diet. Although growth hormone affects calcium metabolism, hormone levels and total body calcium showed no correlation in 15 patients taking levodopa. The findings suggest that a low protein diet benefits patients with Parkinson's disease and with moderate neurologic instability.

L6 ANSWER 69 OF 94 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN

ACCESSION NUMBER: 2003:283664 BIOSIS
DOCUMENT NUMBER: PREV200300283664
TITLE: LIPOPHILIC COMPLEX I INHIBITORS MIMICK ATYPICAL PARKINSONISM IN RATS.
AUTHOR(S): Hglinger, G. U. [Reprint Author]; Champy, P. [Reprint

Author]; Fger, J. [Reprint Author]; Prigent, A. [Reprint Author]; Parain, K. [Reprint Author]; Oertel, W. H.; Hocquemiller, R.; Hirsch, E. C. [Reprint Author]; Ruberg, M. [Reprint Author]
CORPORATE SOURCE: INSERM U289, Hpital de la Salptrire, Paris, France
SOURCE: Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 264.18. http://sfn.scholarone.com. cd-rom.
Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 19 Jun 2003
Last Updated on STN: 19 Jun 2003

AB Reduced mitochondrial complex I activity has been shown in cybrids containing mitochondria from progressive supranuclear palsy (PSP) patients and an epidemiological study in Guadelupe has suggested an association between a PSP-like disease and the consumption of Annonaceae plants containing lipophilic complex I inhibitors.)To assess the consequences of chronic generalized inhibition of complex I, we infused male Lewis rats i.v. for 28d with the lipophilic pesticide rotenone (1.8, 2.2, or 2.5mg/kg/d) or annonacine (1.26, 3.8, or 7.6mg/kg/d), the major acetogenin of Annona muricata, and compared them to vehicle-infused rats. Analysis of rotenone-and annonacine-infused rats gave very similar results. Treated rats showed a significant loss of spontaneous locomotor activity. Striatal dopaminergic fibres and nigral dopaminergic neurones were lost. All animals with nigral lesions also showed loss of striatal DARPP-32-positive projection neurones. Serotoninergic, cholinergic, and noradrenergic systems were also affected. In annonacine-rats astrogliosis was observed. Pronounced neurodegeneration in basal ganglia and brain stem nuclei was evidenced by Bodian silver staining, whereas hippocampus, cerebellum, and cerebral cortex were only moderately affected. Reduced doses of rotenone/annonacine did not confer lesion selectivity for nigral dopaminergic neurons. Our data support the view that a chronic generalized mitochondrial failure may be crucially involved in the development of atypical parkinsonian syndromes such as PSP. GH/PC equal contribution.

L6 ANSWER 70 OF 94 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:495734 BIOSIS
DOCUMENT NUMBER: PREV200100495734
TITLE: Potential benefits of a comprehensive rehabilitation program in Parkinson's disease.
AUTHOR(S): Frey, D. J.; Hernandez, T. D. [Reprint author]; Smith, T. P.; Orent, S. J.; Fleshner, M.
CORPORATE SOURCE: Dept Psychology, Univ Colorado, Boulder, CO, USA
SOURCE: Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 533. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Oct 2001
Last Updated on STN: 23 Feb 2002

AB Parkinson's is a neurodegenerative disease that involves a

progressive destruction of dopaminergic systems leading to severe cognitive and motor impairments. Although the search for effective surgical and pharmacological therapeutic interventions continues, the need for supplemental non-invasive interventions remains great. Thus, the following study tested the efficacy of a supplemental behavioral intervention to slow the decline in physical function in persons with Parkinson's disease. The behavioral intervention was a comprehensive rehabilitation program that involved a combination strength, balance and endurance training administered in a clinical setting (Medically Based Fitness, MBF). 10 patients with Parkinson's disease participated in the program for 2-6 months. Measures included the Berg Balance Assessment, submaximal or endurance until, exhaustion testing, and strength testing. The results were that improvements were found in all measures taken with the largest improvements found two months after training onset. Specifically, improvements in balance (Berg, p=.0001), endurance (p=.003) and strength (knee flexion, knee extension, lat pull, triceps, biceps, ps<.05) compared with pre-training responses were found. Thus, this behavioral intervention resulted in improvements in all measures across time despite the presence of a progressive neurodegenerative disease. Potential mechanisms for these improvements include neural adaptations, strength and cardiorespiratory, workload gains, upregulation of growth hormones, and a dampening of inflammation process.

L6 ANSWER 71 OF 94 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN

ACCESSION NUMBER: 1993:324035 BIOSIS
DOCUMENT NUMBER: PREV199396032385
TITLE: A controlled trial of Lazabemide (RO19-6327) in untreated Parkinson's disease.
AUTHOR(S): Parkinson Study Group
CORPORATE SOURCE: Box 673, Dep. Neurol., Univ. Rochester Med. Cent., 601 Elmwood Ave., Rochester, NY 14642, USA
SOURCE: Annals of Neurology, (1993) Vol. 33, No. 4, pp. 350-356.
CODEN: ANNED3. ISSN: 0364-5134.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Jul 1993
Last Updated on STN: 3 Jan 1995

AB The monoamine oxidase type B inhibitor deprenyl (selegiline) has been demonstrated to delay the emergence of disability in early untreated Parkinson's disease. Lazabemide (RO19-6327) is a short-acting, reversible, highly selective inhibitor of monoamine oxidase type B which, unlike deprenyl, is not metabolized to active compounds. We conducted a randomized, double-blinded clinical trial to assess the short-term tolerability of lazabemide in subjects who had early, untreated Parkinson's disease. Two hundred and one patients were enrolled at 14 centers and randomized to receive 100 mg/day, 200 mg/day, or 400 mg/day of lazabemide or matching placebo. Subjects were followed for 8 weeks including a randomized, double-blinded withdrawal of lazabemide for 2 or 4 weeks. The primary measure of tolerability was the proportion of treated subjects who were able to complete the study on their originally assigned treatment . Clinical features were assessed by the Unified Parkinson's Disease Rating Scale (UPDRS). Lazabemide treatment was as well tolerated as placebo and was not attended by serious adverse experiences. A significant improvement in the activities of daily living component of the rating scale was found after 4 weeks of lazabemide treatment , although other subscale scores did not change significantly. The overall safety and benefits of lazabemide observed in this short-term study justify further long-term investigations to determine if this

monoamine oxidase type B inhibitor can slow the clinical progression of Parkinson's disease.

L6 ANSWER 72 OF 94 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 1986:255944 BIOSIS
DOCUMENT NUMBER: PREV198682010693; BA82:10693
TITLE: EFFECT OF L DOPA ON FRACTURE HEALING OF RAT FIBULA.
AUTHOR(S): LEE J-J [Reprint author]
CORPORATE SOURCE: DEP ORTHOPAEDIC SURGERY, CATHOLIC MED COLLEGE, SEOUL, KOREA
SOURCE: Journal of Catholic Medical College, (1986) Vol. 39, No. 1, pp. 169-178.
CODEN: KTUNAA. ISSN: 0368-7015.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: KOREAN
ENTRY DATE: Entered STN: 21 Jun 1986
Last Updated on STN: 21 Jun 1986
AB Some authors sugggest that endocrine factors would be important in fracture healing, if there are problems of endocrinological basis. Among the humoral substances, growth hormone is well known to be helpful in fracture healing process. But growth hormone is currently expensive and difficult to obtain. In recent years there have been various reports on growth hormone secretion in patients suffering from Parkinson's disease who were treated with L-dopa. The present study was designed to determine the effect of L-dopa on fracture healing. A total of 30 Sprague-Dawley (SD) rats of both sexes were used. The weight of them was from 250 to 300 g. The animals were equally divided into two groups. And each group was assigned to 3 subgroups according to the observation period after the operative transverse fibular fracture (2, 3, 4 weeks, respectively). The experimental animals were dosed 400 mg /Kg/day of L-dopa. The chronologic histology of the healing callus was studied, and for the central callus its histological maturity was evaluated by 5 point scale method after Allen et al. (1980). In the adjacent and peripheral callus region there was no significant difference in its histologic maturity between two groups at each observation time. The postoperative histological maturity of the soft callus was graded 2 at 2 weeks, 3.1 at 3 weeks and 3.9 at 4 weeks, while in the experimental group the grades were 2.4 at 2 weeks, 3.6 at 3 weeks and 4.6 at 4 weeks. More maturity of the soft callus was observed in the experimental group at each observation period. Postoperatively the hard callus was formed in 3 limbs at 3 weeks, and 6 limbs at 4 weeks in control group. But in the experimental group it was formed in 1 limb at 2 weeks, 5 limbs at 3 weeks, and 9 limbs at 4 weeks postoperatively. The hard callus was formed in 9 out of 30 control limbs, but 15 out of 30 experimental limbs totally. Through this study it is inferred that L-dopa which presumably operates its function by stimulating release of growth hormone, accelerates the fracture healing by stimulating endochondral ossification of the central callus.

L6 ANSWER 73 OF 94 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 1982:167166 BIOSIS
DOCUMENT NUMBER: PREV198273027150; BA73:27150
TITLE: EFFECTS OF L DEPRENYL ON HUMAN GROWTH HORMONE SECRETION.
AUTHOR(S): KOULU M [Reprint author]; LAMMINTAUSTA R
CORPORATE SOURCE: DEP OF PHARMACOL, INST OF BIOMED, UNIV OF TURKU, TURKU, FINLAND
SOURCE: Journal of Neural Transmission, (1981) Vol. 51,

No. 3-4, pp. 223-232.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
AB The potentiating effects of L-deprenyl on L-dopa treatment of Parkinson's disease led to the investigation of whether L-deprenyl modifies basal or dopamine-controlled growth hormone secretion. The effects of L-deprenyl on L-dopa-, apomorphine- and L-tryptophan-induced growth hormone (GH) secretion were studied in 13 healthy male volunteers. An acute 10 mg dose of L-deprenyl did not stimulate the basal GH secretion. Short-term L-deprenyl premedication significantly enhanced the L-dopa-stimulated GH release. L-deprenyl premedication did not change the GH response to apomorphine or L-tryptophan. Potentiation of L-dopa-induced GH release by L-deprenyl indicates an increased availability of dopamine at the receptor level without a direct agonistic effect by the drug. L-deprenyl does not change the function of postsynaptic dopamine receptors involved in human GH release.

L6 ANSWER 74 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2002:638136 CAPLUS
DOCUMENT NUMBER: 137:174958
TITLE: Materials and methods for making improved liposome compositions containing amphipathic peptides and proteins
INVENTOR(S): Onyuksel, Hayat; Rubinstein, Israel
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 23 pp., Cont.-in-part of U.S. 6,348,214.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20020114829	A1	20020822	US 2001-995263	20011127 <--
US 6197333	B1	20010306	US 1998-155368	19981218 <--
US 6348214	B1	20020219	US 2000-630699	20000801 <--
WO 2003046145	A2	20030605	WO 2002-US38075	20021127
WO 2003046145	A3	20031016		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2002359511 A1 20030610 AU 2002-359511 20021127
PRIORITY APPLN. INFO.: US 1998-155368 A3 19981218
US 2000-630699 A2 20000801
US 1996-14363P P 19960328
WO 1997-US5161 W 19970328
US 2001-995263 A 20011127
WO 2002-US38075 W 20021127

AB A method for treating autism, multiple sclerosis, eneuresis, Parkinson's disease, amyotrophic lateral sclerosis, brain

ischemia, stroke, cerebral palsy, sleep disorder, feeding disorder and AIDS-associated dementias comprises liposome containing a biol. active amphipathic compound, i.e., peptides and proteins, such as a member of the vasoactive intestinal peptide (VIP)/glucagon/secretin family of peptides including peptide fragments and analogs. Methods for producing the liposome products as well as methods of using the liposome products in therapeutic and diagnostic techniques are also provided. For example, liposomes comprise distearoyl phosphatidylethanolamine covalently bonded to PEG (PEG-DSPE), phosphatidylcholine (PC), and phosphatidylglycerol (PG) in combination with cholesterol (Chol). The lipids and Chol are combined in a PEG-DSPE:PC:PG:Chol molar ratio of 0.5:5:1:3.5.

L6 ANSWER 75 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2002:594822 CAPLUS
DOCUMENT NUMBER: 137:154857
TITLE: Preparation of nicotinamide biaryl derivatives as inhibitors of PDE4 isozymes
INVENTOR(S): Chambers, Robert James; Magee, Thomas Victor; Marfat, Anthony
PATENT ASSIGNEE(S): Pfizer Products Inc., USA
SOURCE: PCT Int. Appl., 224 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002060875	A1	20020808	WO 2001-IB2341	20011206 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2436535	A1	20020808	CA 2001-2436535	20011206 <--
AU 2002220966	A1	20020812	AU 2002-220966	20011206 <--
EP 1355884	A1	20031029	EP 2001-273556	20011206
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
EE 200300360	A	20031215	EE 2003-360	20011206
BR 2001016852	A	20040225	BR 2001-16852	20011206
HU 2004000637	A2	20040628	HU 2004-637	20011206
JP 2004520386	T	20040708	JP 2002-561026	20011206
CN 1518542	A	20040804	CN 2001-823071	20011206
NZ 526453	A	20050128	NZ 2001-526453	20011206
US 20020193612	A1	20021219	US 2002-62813	20020131 <--
US 6649633	B2	20031118		
IN 2003MN00608	A	20050318	IN 2003-MN608	20030617
ZA 2003004894	A	20040624	ZA 2003-4894	20030624
US 20040048903	A1	20040311	US 2003-613988	20030702
US 6953810	B2	20051011		
BG 108038	A	20040730	BG 2003-108038	20030728
NO 2003003397	A	20030919	NO 2003-3397	20030730
MX 2003006887	A	20031113	MX 2003-6887	20030730
PRIORITY APPLN. INFO.:			US 2001-265492P P	20010131

	WO 2001-IB2341	W 20011206
	US 2002-62813	A3 20020131

OTHER SOURCE(S): MARPAT 137:154857
GI

* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA OFFLINE PRINT *

AB The title compds. [I; g = 0-1; j = 0-1; provided that when j = 0, n must be 2; k = 0-1; m = 0-2; n = 1-2; W1 = O, SOt (t = 0-2), NR3; W2 = OCR9R10, or absent; Y = CR1, NOk (k = 0-1); R9, R10 = H, F, CF3, etc.; or R9 and R10 are taken together, but only in the case where m = 1, to form a spiro moiety; R7, R8 have the same meaning as R9, R10 except that one of them must be H; R1, R2 = H, F, Cl, etc.; R3 = H, alkyl, Ph, etc.; R4-R6 = H, F, Cl, etc.; Q1 = Ph, benzodioxyl, etc.; Q2 = biaryl moiety], useful as inhibitors of PDE4 in the treatment of diseases regulated by the activation and degranulation of eosinophils, especially asthma, chronic bronchitis, and chronic obstructive pulmonary disease, were prepared E.g., a multi-step synthesis of the amide II, starting from Me 3-bromobenzoate and 4-formylbenzeneboronic acid, was given. Compds. I showed anti-inflammatory activity at 0.0001 μM to 20.0 μM in whole blood assay for LTE4.

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 76 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN

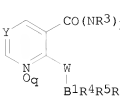
ACCESSION NUMBER:	2002:591707 CAPLUS
DOCUMENT NUMBER:	137:140509
TITLE:	Preparation of nicotinamides and mimetics as inhibitors of phosphodiesterase IV isozymes
INVENTOR(S):	Chambers, Robert J.; Magee, Thomas V.; Marfat, Anthony
PATENT ASSIGNEE(S):	Pfizer Products Inc., USA
SOURCE:	Eur. Pat. Appl., 180 pp. CODEN: EPXXDW
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	3

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
EP 1229034	A1	20020807	EP 2002-250202	20020111	<--
EP 1229034	B1	20050413			
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR					
AT 293109	T	20050415	AT 2002-250202	20020111	
ES 2239203	T3	20050916	ES 2002-250202	20020111	
CA 2369462	A1	20020731	CA 2002-2369462	20020129	<--
MX 2002001141	A	20020918	MX 2002-1141	20020130	<--
US 20020111495	A1	20020815	US 2002-62811	20020131	<--
JP 2002284766	A	20021003	JP 2002-22710	20020131	<--
BR 2002000250	A	20021008	BR 2002-250	20020131	<--
US 20040171798	A1	20040902	US 2004-781062	20040217	
US 7250518	B2	20070731			

PRIORITY APPLN. INFO.:	US 2001-265240P	P	20010131
	US 1997-43403P	P	19970404
	US 1998-105120P	P	19981021

OTHER SOURCE(S): MARPAT 137:140509
GI



I

AB Title compds. [I; p, q = 0, 1; m = 0-2; n = 1, 2; A = CO2R7, CONR9CO2R7, CONR7R9, OP(O)(OH)2, SO3H, acylsulfonamido, etc.; W = O, S, SO, SO2, NR3; Y = N, NO, CR11; R1, R2 = H, F, Cl, cyano, NO2, alkyl, alkynyl, fluoroalkyl, etc.; R3 = H, alkyl, Ph, PhCH2, etc.; R4-R6 = H, F, Cl, alkynyl, cyano, NO2, etc.; R7 = H, (substituted) alkyl, alkenyl, alkynyl; R9 = H, alkyl, cycloalkyl, Ph, PhCH2, pyridyl, etc.; R11 = H, F, Cl, cyano, NO2, alkyl, alkynyl, fluoroalkyl, fluoroalkoxy, etc.; Ra, Rb = H, F, CF3, alkyl, (substituted) cycloalkyl, Ph, PhCH2; B1, B2 = 3-7 membered (hetero)cyclyl, 7-12 membered poly(hetero)cyclyl; pairs of variables may form rings; with provisos], were prepared (no data). Thus, Me 2-[4-[[[2-(benzo[1,3]dioxol-5-yloxy)pyridine-3-carbonyl]amino]methyl]phenyl]-2-methylpropionate was suspended in Me3COH. Aqueous NaOH was added to the suspension, and the reaction mixture was refluxed 1 h to give 2-[4-[[[2-(benzo[1,3]dioxol-5-yloxy)pyridine-3-carbonyl]amino]methyl]phenyl]-2-methylpropionic acid.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 77 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2002:539515 CAPLUS
DOCUMENT NUMBER: 137:88486
TITLE: Fumaric acid amide derivatives with peptides and usage as drugs
INVENTOR(S): Joshi, Rajendra Kumar; Strebel, Hans-Peter
PATENT ASSIGNEE(S): Fumapharm Ag, Switz.
SOURCE: PCT Int. Appl., 20 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002055063	A2	20020718	WO 2002-EP107	20020108 <--
WO 2002055063	A3	20031009		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
DE 10101307	A1	20020801	DE 2001-10101307	20010112 <--

AU 2002219236	A1	20020724	AU 2002-219236	20020108 <--	
AU 2002219236	B2	20060209			
CA 2425599	A1	20030410	CA 2002-2425599	20020108	
EE 200300280	A	20031015	EE 2003-280	20020108	
HU 2003002656	A2	20031128	HU 2003-2656	20020108	
EP 1372634	A2	20040102	EP 2002-729423	20020108	
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR					
JP 2004523511	T	20040805	JP 2002-555797	20020108	
NZ 526100	A	20050429	NZ 2002-526100	20020108	
RU 2290946	C2	20070110	RU 2003-124752	20020108	
BG 107795	A	20040831	BG 2003-107795	20030509	
US 20040038889	A1	20040226	US 2003-433295	20030602	
US 7157423	B2	20070102			
US 20060205659	A1	20060914	US 2006-421083	20060531	
US 7432240	B2	20081007			

PRIORITY APPLN. INFO.:	DE 2001-10101307	A	20010112
	DE 2001-10133004	A	20010706
	WO 2002-EP107	W	20020108
	US 2003-433295	A3	20030602

OTHER SOURCE(S): MARPAT 137:88486

AB The invention relates to the use of fumaric acid amides of general formula R2-CO-(CH)2-CO-R1 wherein R1 represents OR3 or a D- or L- amino acid radical NH-CHR4-COOH bound by an amide bond, wherein R3 is hydrogen, a linear chain or branched, optionally substituted C1-21-alkyl radical Ph radical or a C6-10-aralkyl radical and R4 is a side chain of a natural or synthetic amino acid, and R2 represents a D- or L- amino acid radical bound by an amide bond NH-CHR5-COOH or a peptide radical bound by an amide bond having 2-100 amino acids, wherein R5 is a side chain of a natural or synthetic amino acid, in the production of a medicament for therapy of an autoimmune disease, for use in transplantation medicine, for therapy of mitochondrial diseases and for therapy of NfkappaB mediated diseases.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 78 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER:	2002:172052 CAPLUS
DOCUMENT NUMBER:	136:227997
TITLE:	Sequence of human tyrosine hydroxylase gene promoter and its uses for regulation of gene expression for treatment of Parkinson's disease
INVENTOR(S):	Iacovitti, Lorraine; Kessler, Mark A.
PATENT ASSIGNEE(S):	Thomas Jefferson University, USA
SOURCE:	PCT Int. Appl., 44 pp. CODEN: PIXXD2
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	2
PATENT INFORMATION:	

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002018548	A2	20020307	WO 2001-US26897	20010829 <--
WO 2002018548	A3	20030731		
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR,				

IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2001086888 A 20020313 AU 2001-86888 20010829 <--
PRIORITY APPLN. INFO.: US 2000-228931P P 20000830
WO 2001-US26897 W 20010829

AB The differentiated cells of the adult mammalian central nervous system (CNS) have little or no ability to generate new nerve cells. This inability to produce new nerve cells is a distinct disadvantage when the need to replace lost neurons arises due to injury or disease. The present invention provides the sequence of 10.828 kB of the human tyrosine hydroxylase promoter. This sequence is used to purify dopaminergic cells, thus providing treatment for neurol. diseases or disorders, such as Parkinson's disease, wherein a biol. active tyrosine hydroxylase is limiting or absent.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 79 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2001:208101 CAPLUS
DOCUMENT NUMBER: 134:242675
TITLE: Therapeutic uses of polymers and oligomers comprising γ-hydroxybutyrate
INVENTOR(S): Williams, Simon F.; Martin, David P.
PATENT ASSIGNEE(S): Tepha, Inc., USA
SOURCE: PCT Int. Appl., 18 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001019361	A2	20010322	WO 2000-US25261	20000914 <--
WO 2001019361	A3	20011004		
W: AU, CA, JP				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2383011	A1	20010322	CA 2000-2383011	20000914 <--
CA 2383011	C	20080722		
EP 1212052	A2	20020612	EP 2000-963475	20000914 <--
EP 1212052	B1	20050413		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
JP 2003509366	T	20030311	JP 2001-522995	20000914
US 6623730	B1	20030923	US 2000-661948	20000914
AT 292965	T	20050415	AT 2000-963475	20000914
AU 783104	B2	20050922	AU 2000-74886	20000914
ES 2240163	T3	20051016	ES 2000-963475	20000914

PRIORITY APPLN. INFO.: US 1999-153844P P 19990914
US 2000-182371P P 20000214
WO 2000-US25261 W 20000914

AB Oligomers and polymer compns. are provided which comprise γ-hydroxybutyric acid (GHB) and produce GHB after administration in vivo. Devices for the storage and delivery of these polymers and oligomers are also provided. These oligomers and polymer compns. are useful in a variety of applications. The compns. can be used therapeutically, for example, in the treatment of patients with narcolepsy, chronic schizophrenia, catatonic schizophrenia, atypical psychoses, chronic brain syndrome, neurosis, alcoholism, drug addiction and withdrawal, Parkinson's disease and other neuropharmacol. illnesses, hypertension, ischemia, circulatory collapse,

radiation exposure, cancer, and myocardial infarction. Other uses for the compns. include anesthesia induction, sedation, growth hormone production, heightened sexual desire, anorectic effects, euphoria, smooth muscle relaxation, muscle mass production, and sleep, including rapid eye movement sleep. In a still further embodiment, the oligomers and polymers may be used to produce absence seizures. The GHB oligomer, prepared from poly(γ-hydroxybutyrate) of MW 430,000 by reaction with NaOMe, was digested in the rat providing a sustained release of the monomer, over at least 10 h. The amount of the GHB in the serum was elevated about 3-8 times that of the baseline values over a period of 1-10 h.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 80 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2000:368623 CAPLUS

DOCUMENT NUMBER: 133:13448

TITLE: Adenovirus vector for gene therapy with modified steroid hormone receptor proteins for target gene expression regulation

INVENTOR(S): Burcin, Mark M.; O'Malley, Bert W.; Schiedner, Gudrun; Tsai, Sophia Y.; Kochanek, Stefan

PATENT ASSIGNEE(S): Valentis, Inc., USA

SOURCE: PCT Int. Appl., 64 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000031286	A1	20000602	WO 1999-US26802	19991112 <--
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 20050196751	A1	20050908	US 2001-861181	20010518
PRIORITY APPLN. INFO.:			US 1998-109185P	P 19981120
			WO 1999-US26802	A1 19991112

AB Adenovirus vector for gene therapy with modified steroid hormone receptor proteins as regulator for therapeutic target gene expression regulation are described. To regulate expression of a transferred gene in response to an exogenous compound, a high capacity adenoviral vector devoid of all viral coding sequences with a regulator gene to control a target gene expression in vivo in a selected site and at a desired time are constructed. The regulator GLp65 (a chimeric transactivator) consists of a mutated progesterone receptor-ligand binding domain fused to the GAL4 DNA binding domain and part of the activation domain of the human p65 protein, a component of the NF-κB complex. In the presence of ligand RU486, GLp65 binds to a target gene (hGH) containing the 17-mer GAL4 binding site, resulting in an efficient ligand-inducible transactivation of the target gene. Adenoviral vectors with regulator gene and target gene with or without the insulator sequence (2xHS4, a 5' element of the chicken β-globin domain) are also constructed and tested in animal cells or in transgenic mice. The kinetics of induction and effects of insulator sequence on target gene are studied. Such vectors are capable of achieving high levels and durations

of delivery and expression. The modified regulator protein is capable of distinguishing a hormone agonist from an antagonist and may be modified in the ligand binding domain, the DNA binding domain, and/or the trans-regulatory domain. These regulable adenoviral vectors can be used for potentially diverse applications, ranging from tissue-specific gene expression in transgenic animals to human gene therapy.
REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 81 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1999:127015 CAPLUS
DOCUMENT NUMBER: 130:192740
TITLE: Human glial cell line-derived neurotrophic factor promoters, vectors containing same, and methods of screening compounds therewith
INVENTOR(S): Baecker, Preston Albert; Johnson, Randolph Mellus; Lee, Walter Hom; Verity, Adrian Neil
PATENT ASSIGNEE(S): F. Hoffman-La Roche AG, Switz.
SOURCE: PCT Int. Appl., 100 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
WO 9907843	A1	19990218	WO 1998-EP4620	19980723	<--
W: AU, CA, JP					
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE					
CA 2299586	A1	19990218	CA 1998-2299586	19980723	<--
CA 2299586	C	20070918			
AU 9890679	A	19990301	AU 1998-90679	19980723	<--
AU 737944	B2	20010906			
EP 1002071	A1	20000524	EP 1998-942603	19980723	<--
EP 1002071	B1	20070418			
R: AT, BE, CH, DE, FR, GB, IT, LI, NL, SE, IE					
JP 2001512679	T	20010828	JP 2000-506328	19980723	<--
JP 3761152	B2	20060329			
AT 360066	T	20070515	AT 1998-942603	19980723	
PRIORITY APPLN. INFO.:			US 1997-54812P	P 19970805	
			US 1998-81751P	P 19980414	
			WO 1998-EP4620	W 19980723	

AB The distal and proximal promoters for the human glial cell line-derived neurotrophic factor (GDNF) gene are provided. Regulatory factor binding sites are identified within the promoters, such as sites for epidermal growth factor receptor transcription factor (ETF), early growth response (egr) family, SP1, CREB/ATF, NF-κB, YY- 1, and GC factor (GCF). In addition, constructs comprising a human GDNF promoter and a reporter gene, a vector comprising the construct and a host cell comprising the vector are provided, as is a method for screening compds. capable of modulating the expression of GDNF by stimulating GDNF promoter-directed transcription. The constructs can be used to gene therapy treatment of neurodegenerative diseases such as Parkinson's disease, as well as in screening of pharmaceutical compds.
REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 82 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1999:48056 CAPLUS

DOCUMENT NUMBER: 130:120458
TITLE: Transgenic mice which overexpress neurotrophin-3 (nt-3) and their use in studying and treatment of neurodegenerative disorders.
INVENTOR(S): Albers, Kathryn M.; Davis, Brian M.
PATENT ASSIGNEE(S): University of Kentucky Research Foundation, USA
SOURCE: U.S., 17 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5859311	A	19990112	US 1995-534685	19951127 <--
PRIORITY APPLN. INFO.:			US 1995-534685	19951127

AB The NT-3 growth factor expressing transgenic mice are useful in the study of neurodegenerative disorders of the brain such as Parkinson's syndrome and Alzheimer's disease, of the spinal cord motor neurons such as amyotrophic lateral sclerosis, and for testing drug candidates for the treatment of these diseases. Transgenic mice express increased levels of neurotrophin-3 (NT-3) in epithelium when their ancestors are microinjected with the NT-3 gene. The NT-3 transgene comprises a mouse NT-3 gene linked at its 5'end to a human K14 keratin promoter and enhancer region and at its 3' end to a contiguous 1.8 kbp region of the human growth hormone gene and a polyadenylation signal sequence. The NT-3 transgene was introduced by microinjection to the mouse at embryonic stage. The transgenic mouse is fertile and capable of transmitting the NT-3 transgene to its offspring. The NT-3 responsive neurons of the transgenic mouse were rescued from programmed cell death during mouse development. The NT-3 transgene encoding mice revealed an increase in sensory neurons that express trk C receptor and had larger Merkel cell sensory units in the skin.
REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 83 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1997:326866 CAPLUS
DOCUMENT NUMBER: 126:308798
ORIGINAL REFERENCE NO.: 126:59765a,59768a
TITLE: Chimeric DNA-binding/DNA methyltransferase nucleic acid and polypeptide and their uses
INVENTOR(S): Bestor, Timothy H.
PATENT ASSIGNEE(S): Trustees of Columbia University in the City of New York, USA; Bestor, Timothy H.
SOURCE: PCT Int. Appl., 97 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9711972	A1	19970403	WO 1996-US15576	19960927 <--
W: AU, CA, JP, MX, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9673781	A	19970417	AU 1996-73781	19960927 <--
US 20020188103	A1	20021212	US 1998-51013	19981009 <--
PRIORITY APPLN. INFO.:			US 1995-4445P P	19950928
			US 1996-594866 A2	19960131

AB The present invention provides a chimeric protein which comprises a mutated DNA methyltransferase portion and a DNA binding protein portion that binds sufficiently close to a promoter sequence of a target gene (which promoter sequence contains a methylation site) to specifically methylate the site and inhibit activity of the promoter and thus inhibit expression of the target gene. This invention also provides for a method for inhibiting the expression of a target gene which includes contacting a promoter of the target gene with the chimeric protein, so as to specifically methylate the promoter sequence of the target gene thus inhibiting expression of the target gene.

L6 ANSWER 84 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1997:169088 CAPLUS
DOCUMENT NUMBER: 126:182303
ORIGINAL REFERENCE NO.: 126:35101a,35104a
TITLE: Transgenic mice which overexpress nerve growth factor
INVENTOR(S): Albers, Kathryn M.; Davis, Brian M.
PATENT ASSIGNEE(S): University of Kentucky Research Foundation, USA
SOURCE: U.S., 19 pp., Cont. of U.S. Ser. No. 131,424, abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5602309	A	19970211	US 1995-438122	19950508 <--
PRIORITY APPLN. INFO.:			US 1993-131424	B1 19931004

AB Transgenic mice are provided that express increased levels of nerve growth factor (NGF) in the epidermis and other stratified, keratinized epithelium. A 797-bp cDNA fragment encoding the NGF short transcript was ligated into a K14-hGH plasmid containing 2.1 kbp of 5'-upstream sequence of the human K14 keratin gene and a 1.8-kbp intron containing sequence from the human growth hormone gene. The hGH sequence serves to upregulated expression of the transgene and provides a polyadenylation signal. The plasmid is introduced into a fertilized mouse embryo by microinjection. Transgenic mice have a phenotype characterized by hyperinnervation of the skin, hypertrophy of the trigeminal ganglion, and enlargement of the superior cervical sympathetic and dorsal root ganglia when compared to a normal mouse. The nerve growth factor expressing transgenic mice are useful in the study of neurodegenerative disorders of the brain such as Parkinson's syndrome and Alzheimer's disease and for testing for drug candidates for the treatment of these diseases.
REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 85 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1995:723143 CAPLUS
DOCUMENT NUMBER: 123:102794
ORIGINAL REFERENCE NO.: 123:18031a,18034a
TITLE: Pharmaceutical compositions and use thereof for treatment of neurological diseases and etiologically related symptomatology.
INVENTOR(S): Shapiro, Howard K.
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 155 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9501096	A1	19950112	WO 1994-US7277	19940628 <--
W: AU, CA, JP				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 5668117	A	19970916	US 1993-62201	19930629 <--
AU 9472144	A	19950124	AU 1994-72144	19940628 <--
AU 692454	B2	19980611		
EP 707446	A1	19960424	EP 1994-921405	19940628 <--
R: DE, FR, GB, IT				
JP 08512055	T	19961217	JP 1994-503597	19940628 <--

PRIORITY APPLN. INFO.:	US 1993-62201	A	19930629
	US 1991-660561	B1	19910222
	US 1993-26617	B2	19930223
	WO 1994-US7277	W	19940628

AB Pharmaceutical compns. for treatment of several neurol. diseases and pathophysiol.-related symptomol. in other body tissues, including peripheral neuropathies, secondary symptomol. of diabetes, Alzheimer's disease, Parkinson's disease, alc. polyneuropathy and age-onset symptomol., as well as analogous veterinary diseases, are disclosed. Spurious pathol. chemical crosslinking of normal intracellular structures is a fundamental aspect of these neurol. diseases. Covalent bond crosslinking of protein and lipid subcellular elements appear to underlie the formation of polymerized aggregates of neurofilaments and other structural proteins, and lipofuscin. Pharmacol. intervention in some neurol. diseases using water-soluble, small mol. weight primary amines or their derivs. as oral therapeutic agents, may compete with cellular protein and lipid amine groups for reaction with disease-induced carbonyl-containing aliphatic and aromatic hydrocarbons. Primary pharmacol. agents include 4-aminobenzoic acid and derivs. thereof to facilitate kidney recognition and removal. This invention also includes oral use of nonabsorbable polyamine polymers and amine-related co-agents, such as chitosan, to covalently bind and sequester potentially toxic carbonyl compds. present in the diet, oral use of known antioxidant co-agents and related nutritional factors and use of the primary agent and co-agents in combination with known medicaments for treatment of these neurol. diseases.

REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 86 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1994:646342 CAPLUS
DOCUMENT NUMBER: 121:246342
ORIGINAL REFERENCE NO.: 121:44735a,44738a
TITLE: Arginine derivatives as quisqualate antagonists for treatment of neurological disorders
INVENTOR(S): Huth, Andreas; Loeschmann, Peter-Andreas; Turski, Lechoslaw
PATENT ASSIGNEE(S): Schering A.-G., Germany
SOURCE: Ger. Offen., 5 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

DE 4311806 A1 19941006 DE 1993-4311806 19930403 <--
PRIORITY APPLN. INFO.: DE 1993-4311806 19930403
AB The L- or D-arginine derivs. R3NHC(:Y)NR2(CH2)nCH(NHR1)C(:O)X (R1 = H, C1-6 alkyl, C1-5 alkanoyl, CO2CH2Ph, CO2CMe3; R2, R3 = H, C1-6 alkyl, NO2; X = NHR4, OR5; Y = O, NR6; n = 2-4; R4-R6 = H, C1-6 alkyl) are quisqualate (glutamate) antagonists useful for treatment of neurol., psychiatric, and neuroendocrine disorders, including Parkinson's and Alzheimer's diseases, Huntington's chorea, and amyotrophic lateral sclerosis. Thus, D-arginine-HCl (17 mg/kg injected into the left cerebral ventricle) increased by 50% the dose of quisqualate needed to induce clonic spasms in mice.

L6 ANSWER 87 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1994:226984 CAPLUS
DOCUMENT NUMBER: 120:226984
ORIGINAL REFERENCE NO.: 120:40121a,40124a
TITLE: Compositions of oral nondissolvable matrixes for transmucosal administration of medicaments
INVENTOR(S): Stanley, Theodore H.; Hague, Brian
PATENT ASSIGNEE(S): University of Utah Research Foundation, USA
SOURCE: U.S., 20 pp. Cont.-in-part of U.S. 4,863,737.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 9
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5288498	A	19940222	US 1989-403752	19890905 <--
US 4671953	A	19870609	US 1985-729301	19850501 <--
EP 487520	A1	19920603	EP 1989-909497	19890816 <--
EP 487520	B1	19950412		
R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
JP 05501539	T	19930325	JP 1989-504878	19890816 <--
JP 2801050	B2	19980921		
AU 641127	B2	19930916	AU 1989-40704	19890816 <--
AT 120953	T	19950415	AT 1989-909497	19890816 <--
CA 1338978	C	19970311	CA 1989-609378	19890824 <--
AU 9050352	A	19910408	AU 1990-50352	19890905 <--
AU 645966	B2	19940203		
EP 493380	A1	19920708	EP 1990-902584	19890905 <--
EP 493380	B1	19971029		
R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
US 5132114	A	19920721	US 1989-402881	19890905 <--
JP 05501854	T	19930408	JP 1990-502779	19890905 <--
CA 1339075	C	19970729	CA 1989-610329	19890905 <--
AT 159658	T	19971115	AT 1990-902584	19890905 <--
CA 2066403	A1	19910306	CA 1990-2066403	19900803 <--
CA 2066403	C	19980414		
WO 9103236	A1	19910321	WO 1990-US4369	19900803 <--
W: AU, CA, JP, NO				
RW: AT, BE, CH, DE, DK, ES, FR, GB, IT, LU, NL, SE				
AU 9063371	A	19910408	AU 1990-63371	19900803 <--
AU 642664	B2	19931028		
EP 490944	A1	19920624	EP 1990-913359	19900803 <--
EP 490944	B1	19960529		
R: AT, BE, CH, DE, DK, ES, FR, GB, IT, LI, LU, NL, SE				
JP 05500058	T	19930114	JP 1990-512483	19900803 <--
JP 2749198	B2	19980513		
AT 138562	T	19960615	AT 1990-913359	19900803 <--
ES 2089027	T3	19961001	ES 1990-913359	19900803 <--

NO 9200565	A	19920213	NO 1992-565	19920213	<--
NO 304056	B1	19981019			
DK 9200193	A	19920214	DK 1992-193	19920214	<--
DK 175779	B1	20050214			
NO 9200858	A	19920304	NO 1992-858	19920304	<--
NO 9200855	A	19920410	NO 1992-855	19920304	<--
NO 9200854	A	19920427	NO 1992-854	19920304	<--
DK 9200300	A	19920505	DK 1992-300	19920305	<--
DK 175773	B1	20050214			
AU 9460697	A	19940623	AU 1994-60697	19940427	<--
US 5855908	A	19990105	US 1994-339655	19941115	<--

PRIORITY APPLN. INFO.:

US 1985-729301	A2	19850501
US 1987-60045	A2	19870608
EP 1989-909497	A	19890816
WO 1989-US3518	W	19890816
US 1989-403752	A	19890905
WO 1989-US3801	A	19890905
WO 1990-US4369	A	19900803
US 1993-152414	B1	19931112

AB Compns. and methods of manufacture for producting a medicament composition capable of absorption through the mucosal tissues of the mouth, pharynx, and esophagus are disclosed. The present invention relates to such compns. and methods which are useful in administering lipophilic and nonlipophilic drugs in a dose-to-effect manner such that sufficient drug is administered to produce precisely a desired effect. The invention also relates to manufacturing techniques that enable therapeutic agents to be incorporated into nondissolvable drug containment matrixes which are capable of releasing the drug within a patient's mouth. An appliance or holder is preferably attached to the drug containment matrix. Employing the present invention the drug may be introduced into the patient's bloodstream almost as fast as through injection, and much faster than using the oral administration route, while avoiding the neg. aspects of both of these methods. The nondissolvable drug containment matrix may include permeation enhancers to increase the drug adsorption by the mucosal tissues of the mouth. The matrix composition may also include pH buffering agents to modify the saliva pH thereby increasing the absorption of the drug through the mucosal tissues. Figures show views of some dosage forms.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 88 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER:	1994:226981 CAPLUS
DOCUMENT NUMBER:	120:226981
ORIGINAL REFERENCE NO.:	120:40120h,40121a
TITLE:	Compositions of oral dissolvable medicaments
INVENTOR(S):	Stanley, Theodore H.; Hague, Brian
PATENT ASSIGNEE(S):	University of Utah, USA
SOURCE:	U.S., 22 pp. Cont.-in-part of U.S. 4,863,737. CODEN: USXXAM
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	9

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
US 5288497	A	19940222	US 1989-403751	19890905	<--
US 4671953	A	19870609	US 1985-729301	19850501	<--
EP 487520	A1	19920603	EP 1989-909497	19890816	<--
EP 487520	B1	19950412			

R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE

JP 05501539	T	19930325	JP 1989-504878	19890816 <--
JP 2801050	B2	19980921		
AU 641127	B2	19930916	AU 1989-40704	19890816 <--
AT 120953	T	19950415	AT 1989-909497	19890816 <--
CA 1338978	C	19970311	CA 1989-609378	19890824 <--
AU 9050352	A	19910408	AU 1990-50352	19890905 <--
AU 645966	B2	19940203		
EP 493380	A1	19920708	EP 1990-902584	19890905 <--
EP 493380	B1	19971029		

R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE

US 5132114	A	19920721	US 1989-402881	19890905 <--
JP 05501854	T	19930408	JP 1990-502779	19890905 <--
CA 1339075	C	19970729	CA 1989-610329	19890905 <--
AT 159658	T	19971115	AT 1990-902584	19890905 <--
CA 2066423	A1	19910306	CA 1990-2066423	19900803 <--
CA 2066423	C	19980414		
WO 9103237	A1	19910321	WO 1990-US4384	19900803 <--

W: AU, CA, JP, NO

RW: AT, BE, CH, DE, DK, ES, FR, GB, IT, LU, NL, SE

AU 9062877	A	19910408	AU 1990-62877	19900803 <--
AU 645265	B2	19940113		
EP 490916	A1	19920624	EP 1990-912733	19900803 <--
EP 490916	B1	19951018		

R: AT, BE, CH, DE, DK, ES, FR, GB, IT, LI, LU, NL, SE

JP 05503917	T	19930624	JP 1990-512229	19900803 <--
EP 630647	A1	19941228	EP 1994-111352	19900803 <--
EP 630647	B1	19990303		

R: AT, BE, CH, DE, DK, ES, FR, GB, IT, LI, LU, NL, SE

AT 129148	T	19951115	AT 1990-912733	19900803 <--
ES 2077686	T3	19951201	ES 1990-912733	19900803 <--
AT 177007	T	19990315	AT 1994-111352	19900803 <--
ES 2133448	T3	19990916	ES 1994-111352	19900803 <--
NO 9200565	A	19920213	NO 1992-565	19920213 <--
NO 304056	B1	19981019		
DK 9200193	A	19920214	DK 1992-193	19920214 <--
DK 175779	B1	20050214		
NO 9200857	A	19920406	NO 1992-857	19920304 <--
NO 304348	B1	19981207		
NO 9200855	A	19920410	NO 1992-855	19920304 <--
NO 9200854	A	19920427	NO 1992-854	19920304 <--
DK 9200300	A	19920505	DK 1992-300	19920305 <--
DK 175773	B1	20050214		
AU 9455218	A	19940428	AU 1994-55218	19940218 <--
AU 668004	B2	19960418		
AU 9460697	A	19940623	AU 1994-60697	19940427 <--
US 5824334	A	19981020	US 1996-636828	19960419 <--
US 5783207	A	19980721	US 1997-795359	19970204 <--
US 5785989	A	19980728	US 1997-822560	19970319 <--

PRIORITY APPLN. INFO.:

US 1985-729301	A2	19850501
US 1987-60045	A2	19870608
EP 1989-909497	A	19890816
WO 1989-US3518	W	19890816
US 1989-403751	A	19890905
WO 1989-US3801	A	19890905
EP 1990-912733	A3	19900803
WO 1990-US4384	A	19900803
US 1993-152396	B1	19931112
US 1994-333233	B2	19941102
US 1995-439127	B1	19950511

AB Compns. and methods of manufacture for producing a medicament composition capable of

absorption through the mucosal tissues of the mouth, pharynx, and esophagus are disclosed. The present invention relates to such compns. and methods which are useful in administering lipophilic and nonlipophilic drugs in a dose-to-effect manner that sufficient drug is administered to produce precisely a desired effect. The invention also relates to a manufacturing technique that enables a therapeutic agent or drug to be incorporated into a flavored dissolvable matrix. An appliance or holder is preferably attached to the dissolvable matrix. Employing the present invention the drug may be introduced into the patient's bloodstream almost as fast as through injection, and much faster than using the oral administration route, while avoiding the neg. aspects of both of these methods. The present invention achieves these advantages by incorporating the drug into a carbohydrate, fat, protein, wax, or other dissolvable matrix composition The dissolvable matrix may include permeation enhancers to increase the drug absorption by the mucosal tissues of the mouth. The matrix composition may also include pH buffering agents to modify the salival pH thereby increasing the absorption of the drug through the mucosal tissue. Methohexital sodium was incorporated into a dissolvable matrix including citric acid; ribotide; Compritol 888; aspartame; vanilla, wild cherry, and peppermint microcapsules; compressible sugar; and maltodextrin.

REFERENCE COUNT: 75 THERE ARE 75 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 89 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1992:549301 CAPLUS
DOCUMENT NUMBER: 117:149301
ORIGINAL REFERENCE NO.: 117:25869a,25872a
TITLE: Use of cytokine or growth hormone as protective agent against reactive oxygen species
INVENTOR(S): Wong, Grace H. W.
PATENT ASSIGNEE(S): Genentech, Inc., USA
SOURCE: PCT Int. Appl., 27 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9207578	A1	19920514	WO 1991-US7759	19911021 <--
W: AU, CA, JP				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE				
CA 2092718	A1	19920426	CA 1991-2092718	19911021 <--
CA 2092718	C	20070807		
AU 9189412	A	19920526	AU 1991-89412	19911021 <--
AU 661463	B2	19950727		
EP 554381	A1	19930811	EP 1991-920537	19911021 <--
EP 554381	B1	19960117		
EP 554381	B2	20000426		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE				
JP 06503320	T	19940414	JP 1992-500764	19911021 <--
AT 133073	T	19960215	AT 1991-920537	19911021 <--
JP 2002179590	A	20020626	JP 2001-356092	19911021 <--

PRIORITY APPLN. INFO.: US 1990-602850 A 19901025
JP 1992-500764 A3 19911021
WO 1991-US7759 W 19911021

AB Tumor necrosis factor-α or -β, growth hormone , interleukin-1 (IL-1), or D-factor are useful as protective agents in compns. for protection, inhibition, and prevention of the deleterious effects of reactive O species. Also described are

treatment of transplantable tissues and organs, perfusion solns., and preparation of perfused, excised tissue. Tumor necrosis factor (TNF) protected isolated rat hearts from damage mediated by ischemia and reperfusion. TNF or IL-1 alone were effective in preventing hyperoxia-caused death in rats; growth hormone and D-factor were less effective when used alone, but acted synergistically with TNF and/or IL-1 in protecting the animals.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 90 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1990:605514 CAPLUS
DOCUMENT NUMBER: 113:205514
ORIGINAL REFERENCE NO.: 113:34577a,34580a
TITLE: Human growth hormone in treating human central nervous system diseases
INVENTOR(S): Aroonsakul, Chaovanee
PATENT ASSIGNEE(S): USA
SOURCE: U.S., 8 pp. Cont.-in-part of U.S. 4,791,099.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
US 4898856	A	19900206	US 1988-156242	19880216	<--
US 4791099	A	19881213	US 1984-666254	19841029	<--
US 4727041	A	19880223	US 1986-852645	19860416	<--
AT 157546	T	19970915	AT 1988-100233	19880111	<--
ES 2109914	T3	19980201	ES 1988-100233	19880111	<--
JP 09216837	A	19970819	JP 1997-38275	19880222	<--
US 4897389	A	19900130	US 1989-293134	19890103	<--
US 4902680	A	19900220	US 1989-293017	19890103	<--
US 4898857	A	19900206	US 1989-293132	19890203	<--

PRIORITY APPLN. INFO.:

US 1984-666254	A2	19841029
US 1986-852645	A2	19860416
EP 1988-100233	A	19880111
US 1988-156242	A1	19880216
JP 1988-39323	A3	19880222

AB Human growth hormone (somatotropin), 1-20 mg/kg, is used for treating human central nervous system diseases: Alzheimer's disease, multiple sclerosis, cerebrovascular accidents, Parkinson's disease, senile dementia, cerebral atrophy, cerebellar atrophy, senile tremor, and essential tremor (no data).

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 91 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1988:564285 CAPLUS
DOCUMENT NUMBER: 109:164285
ORIGINAL REFERENCE NO.: 109:27123a,27126a
TITLE: Non-chromaffin tissue plus nerve growth factor reduces experimental parkinsonism in aged rats
AUTHOR(S): Pezzoli, Gianni; Fahn, Stanley; Dwork, Andrew; Truong, Daniel D.; De Yebenes, Justo G.; Jackson-Lewis, Vernice; Herbert, Joseph; Cadet, Jean Lud
CORPORATE SOURCE: Neurol. Inst., New York, NY, 10032, USA
SOURCE: Brain Research (1988), 459(2), 398-403
CODEN: BRREAP; ISSN: 0006-8993

DOCUMENT TYPE: Journal
LANGUAGE: English
AB The mechanisms by which intrastriatal grafts of chromaffin tissue alleviate the signs of chemical and exptl. parkinsonism remain elusive. In conjunction with the intraventricular infusion of nerve growth factor (NGF), ventricular grafts of either nonchromaffin (adipose tissue of sciatic nerve) or adrenal medullary tissue were equally effective in decreasing apomorphine-induced circling in rats whose substantia nigra have been permanently lesioned with 6 -hydroxydopamine. These treatments were much more effective than implantation of the adrenal medulla without NGF. In addition, the effects persisted indefinitely, though at a decreased level, after discontinuation of the NGF infusion. Apparently, trophic factors may be crucial to the beneficial effects of intracerebral transplanted tissues.

L6 ANSWER 92 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1981:189907 CAPLUS
DOCUMENT NUMBER: 94:189907
ORIGINAL REFERENCE NO.: 94:31055a,31058a
TITLE: Electroconvulsive therapy and receptor sensitivity
AUTHOR(S): Modigh, K.; Balldin, J.; Eden, S.; Granerus, A. K.; Waalinder, J.
CORPORATE SOURCE: Psychiatric Res. Cent., St. Jorgen's Hosp., Swed.
SOURCE: Acta Psychiatrica Scandinavica, Supplementum (1981), 290(Recent Adv. Treat. Depression), 91-9
CODEN: ASSUA6; ISSN: 0065-1591
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Administration to rats of 1 electroconvulsion daily for 7 days (ECS + VII) resulted in enhanced behavioral responses to dopamine (DA) agonists and enhanced growth hormone (GH) secretion after treatment with reserpine followed by the DA agonist apomorphine and the noradrenaline (NA) agonist clonidine. The GH response to reserpine followed by clonidine alone was unaffected by pretreatment with ECS + VII. The enhancements, which in animals persist for .gtoreq.10 days, indicate increased responsiveness to DA-sensitive structures. The treatment is assumed to engage structures connected to the DA receptors rather than the receptors themselves. The GH response was studied in depressed patients before and after electroconvulsive therapy (ECT) and twice also in a control group, where the subjects received no treatment between the 2 investigations. ECT induced no unitary change in GH responses. The intraindividual variation was greater in the ECT-treated patients than in the controls. Eight parkinsonian patients with partial therapy resistance to L-DOPA were administered ECT during maintenance of their L-DOPA therapy. Six of 8 patients improved with respect to their extrapyramidal symptoms. The improvement after ECT was significantly correlated to the duration of the L-DOPA therapy but not to the degree of mental depression. The results indicate that changes, related to DA receptors, also develop when ECT is administered clin.

L6 ANSWER 93 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1973:543001 CAPLUS
DOCUMENT NUMBER: 79:143001
ORIGINAL REFERENCE NO.: 79:23161a,23164a
TITLE: Preliminary trials on the effects of L-Dopa and L-Dopa-decarboxylase inhibitor combinations on the secretion of some hypophyscal hormones of Parkinson's disease

AUTHOR(S): De Divitiis, E.; Cerillo, A.; Tata, M. R.; Carella, C.; Lombardi, G.; Criscuolo, T.; Oliver, Ch.; Jaquet, Ph.
CORPORATE SOURCE: Naples, Italy
SOURCE: Rivista di Farmacologia e Terapia (1973), 4(1), 101-11
CODEN: RVFTBB; ISSN: 0302-1750
DOCUMENT TYPE: Journal
LANGUAGE: Italian
AB The addition of a decarboxylase inhibitor to L-dopa (I) [59-92-7] during the therapy of Parkinsonism did not significantly potentiate the effects of I on hormone secretion. The study was performed in 2 patients with Parkinson's disease treated with single oral administration of L-dopa, L-dopa-Ro 8-0576/12 mixture or L-dopa-Ro 8-0576/7 mixture The blood cortisol [50-23-7] level was increased in a dose-related manner by L-dopa (500-2000 mg/dose); with the highest dose, the increase was more prolonged. When L-dopa (100-400 mg) was combined with Ro 8-0576/12 [39430-03-0] (25 mg) or Ro 8-0576/7 [39430-02-9] (50 or 100 mg), no changes in the cortisol level were seen. The blood ACTH [9002-60-2] level responded similarly to the cortisol level after treatment, while the response of the blood growth hormone [9002-72-6] level was similar with respect to L-dopa treatment and variable with respect to treatment with the mixts.

L6 ANSWER 94 OF 94 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1973:461763 CAPLUS
DOCUMENT NUMBER: 79:61763
ORIGINAL REFERENCE NO.: 79:9927a,9930a
TITLE: Failure of amantadine to modify serum growth hormone and insulin levels
AUTHOR(S): Cavagnini, F.; Pontiroli, A. E.; Raggi, U.; Peracchi, M.; Malinverni, A.
CORPORATE SOURCE: Inst. Clin. Med., Univ. Milano, Milan, Italy
SOURCE: Experientia (1973), 29(5), 573
CODEN: EXPEAM; ISSN: 0014-4754
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Amantadine-HCl (I-HCl) [665-66-7] (300 mg, oral or i.v.) did not significantly alter serum growth hormone [9002-72-6] or insulin [9004-10-8] or blood glucose levels in healthy human subjects, but increased the plasma free fatty acid level. The results are discussed in relation to the potentiating effect of I in the treatment of Parkinsonism by dopa.

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